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THE NATURE OF THE OBJECTIVE CAUSE OF SENSATION.—Part II. TASTE.

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Hobbes (subsequently Hartley and many English psychologists) viewed sensation as the result of a pressure or action of an external particle of matter upon the appropriate sense-organ, and a transmission along a nerve to the brain. Those multitudinous feelings of sight and hearing, of taste and smell, of which we are conscious, were all, according to this thinker, the result of transmissions to the brain excited from without; nor is this view without modern scientific support.

It is not one of the newest triumphs of microscopical research to show that the eye, the ear, the nose, and the tongue are all beset with little cellular particles, attached to the sensory nerves of those parts, and extremely sensitive to the "pressures" or "actions" of external particles of matter. Let us call these the sensitive cells or end-organs of the body, understanding by this term that they are formed on the external ends of the nerves. These end-organs are all very much alike in shape and general appearance, it matters not from which sense-organ you take them for examination. In all cases the part attached to the nerve is thick, granular like ground glass, and with a kernel or nucleus; the outer and more projecting part is drawn out into a rod or hair. It is the

style or hair which, directly stimulated from without the body, excites the nerve through the inner segment, sending an impulse to the brain, and giving rise to a sensation.

No doubt an anatomist could, by careful inspection, tell at once the end-organs, taken from the retina of the eye, from those of the ear, or the nose, yet they are all formed, as it were, on the same plan.

No one has insisted more emphatically than Mr. Herbert Spencer that these end-organs have all come from common parentage, hence, in part, their similarity. We are to look, in fact, if we wish to understand their nature aright, upon these different end-organs as arising, by slow development, during the evolution of the animal world, from a layer of simple cells—ectodermic—found covering the bodies of our more primitive ancestors. Most of these ectodermic cells remain as the cells of the skin, but some are changed into the end-organs of the special senses we are discussing.

Now this development, Mr. Herbert Spencer would say, is in part the result of the action of the surrounding forces or the environment upon the organism itself. These acting for many generations have produced changes which depend upon the nature of the organism and of its environment.

That motion of the ether which travels with such velocity and which, striking the eye, sets up an impulse in the optic nerve, and on reaching the brain produces a sensation we call light, is a vibratory motion.

That motion of the air—not of the same velocity as light—which, striking the ear, produces a sensation we call sound, when the impulse it sets up reaches the brain along the auditory nerve, is also a *vibration*.

No one doubts at the present time that all material particles are in constant motion of a vibratory nature. The molecules of a gas are vibrating; so are those of a liquid and a solid. The particles of salt in the sea, or the sugar in a cup of tea, are in constant motion; so are the molecules of quinine and quassia, the odorous particles of musk and of the rose.

We see then that the body is surrounded and acted upon by various forms of matter in vibratory motion. The environments which have developed the special organs out of

the more simple ectodermic cells are moving particles of matter, and they have in all the sense-organs produced the end-organs before alluded to—armed with little rods or styles, the identical structures capable of being to an extraordinary degree affected by motion.

With these general qualities common to the environments of all the special senses, which senses are built up so essentially on the same type, we shall expect to find very striking analogies between the modes of production of the different senses. Thanks to the labours of Young, Maxwell and Helmholtz, we know a good deal about the way in which the organism is stimulated by light and sound, but we know almost nothing about the production of taste and smell. Indeed this matter has as yet received so little attention that scarcely anything is known to the specialist which is not the intellectual property of every one.

The following paper is an investigation into the nature of the production of taste sensations; the question of the production of smell sensations I shall discuss in a subsequent paper.

Let us take for our guide this unity of plan so evident from a study of the microscopical anatomy of the sense-organs; let us study, first the production of sight and hearing, and then endeavour to find analogous ways in which it may be possible to account for the production of taste.

The end-organs of the retina are stimulated by the vibrations of that rarefied matter called ether, in a way as yet not clearly understood. These vibrations differ very much from one another in rapidity or pitch, some are so slow—the ultra-red rays of the spectrum—that they produce no sensation at all. Some again—the ultra-violet—produce no sensation, because they are too rapid. Between these two extremes we find a large number of rays of the visible spectrum—which when they fall upon the eye cause a sensation of light and colour. Now it is found that the kind of colour, or, as we may call it, the quality of the sensation, depends directly upon the pitch of the vibration and nothing else. A somewhat slow vibration causes a sensation of red, a more rapid one a sensation of orange, and so on. It is seldom in nature that the eye is stimulated by vibrations of

only one pitch; thus I have in front of me a book that has a cover of a crimson colour, and I know by experiment that this crimson sensation is only produced when the eye receives at the same time from the same object at least two sets of vibrations. The vibrations that produce the sensations of crimson, scarlet, purple, lilac, &c., are all complex; that is, they are combinations of two or more sets of simple vibrations.

We see then that it is the *kind of vibration* which determines the quality of the sensation in the sense of sight, and that there are differences in the vibratory ether outside the body to correspond with the almost limitless colour variations of which we are sensible.

Let us turn to hearing. If we place a long piece of steel in a vice, and alter its length at will, we shall find that on striking it with a rod it will vary in the rapidity of its vibrations, and various sound sensations will be produced.

If it vibrate slowly, some ten times a second, we shall hear nothing, and if it vibrate very rapidly indeed we shall hear nothing; but, as in the case of sight, we shall find varying sound sensations produced within these extreme limits. The quicker the vibration, the shriller the sound.

In nature, observe the analogy with the ether vibration; sonorous vibrations are seldom simple. From a flute, or a violin, we have a bundle of vibrations striking the ear when a note is sounded. The vibration is complex—generally consisting of a slow vibration called a fundamental tone, and a series of overtones harmonically related to it. The sound of a flute is quite different from that of a violin, even when the same note in the musical scale is sounded in both cases. The reason is that the bundles of vibrations are different, the overtones differing in intensity and arrangement. We see therefore that the *kind of vibration* falling on the ear determines the quality of the sound produced. Both in sight and hearing the sensations will depend upon the pitch or rapidity of the vibration if it is a simple one; or if it is complex, upon the pitch and arrangement of the component simple vibrations. We may say then that the kind or character of the vibratory motion determines the quality of the sensation in both cases. A knowledge of these facts, together with the known similarity

in structure of the end-organs of all the senses, will naturally lead an investigator who wishes to study taste or smell production to search for analogous conditions in these senses also.

In sound and light we have motions varying to an almost limitless degree in their kind or character. Have we motions of the same or similar nature in rapid solution?

As we have seen, matter in the liquid state is in motion, the molecules of sugar in water are in motion, and what is more, the motion is characteristic of the substance. The motion of salt is different from the motion of sugar, and the motion of sugar from the motion of quinine. We have then a field for inquiry open to us, for if we can connect definite taste sensations with definite motions we shall have the analogy we seek. Let us proceed to this inquiry. We know that by increasing the weight of a tuning-fork, and also by increasing its bulk, we can cause it to vibrate more slowly, and the same law has been found to hold good for ultimate atoms and molecules, the proof of which will be given later. Is there then any relation between taste sensations and the molecular height of sapid substances?

If we take the molecular weights of acid substances, we shall find in the case of hydrochloric acid a weight of 36·5, in the case of sulphuric acid 98. Lithium chloride, molecular weight 42·5, and potassium sulphate, molecular weight 174, are saline. Many other examples might be adduced from bitter and sweet compounds, to demonstrate that we cannot tell by the molecular weight alone anything about the taste of a substance. In the above example, an acid may have a lower or a higher molecular weight than a salt.

We shall see hereafter that molecular weight is not without its importance, and even at this stage of our inquiry it may be well to allude to a series of facts in this relationship. Bodies which have the greatest molecular weight of any, such as albumens, albuminoids, starches, gums, &c., are totally without taste; nor need it be urged that a ready explanation of this is at hand. No doubt many of these substances are not readily soluble in water, and in consequence they will not readily permeate the viscous layer of saliva covering the tongue and the surface of the gustatory end-organs. Some

albumens, such as serum and egg albumen, are fairly soluble, and they may all be converted by appropriate ferments into allied products called peptones, which are soluble and readily diffusible, and nevertheless devoid of taste. In like manner, starch may be converted into dextrin, a substance readily diffusible in water, and tasteless.

We find also among the list of tasteless bodies all those of very small molecular weight. Water, molecular weight 18, is an example. The simple gases dissolved in water are tasteless, but when we examine substance with a molecular weight over 30, we find that when dissolved in water it is capable of producing some taste or other. Sulphurated hydrogen, molecular weight 34, is very slightly acid to the taste; hydrochloric acid, molecular weight 36·5, is very acid; and nitrogen monoxide, molecular weight 46, is sweet. It is true that the monatomic alcohols, such as ordinary commercial alcohol, have molecular weights over 34, and are tasteless, so that the same rule does not apply to all the carbon compounds; but even here we are struck with the fact, that these alcohols are the lowest of a series, all the polyatomic and more complex alcohols being sapid and sweet in character.

There seems then to be some analogy between taste on the one hand, and sight on the other. The substances of lowest molecular weight, and therefore of most rapid vibration, like the ultra-violet rays of the spectrum, are incapable of producing sensation. In like manner, there are substances of very great molecular weight, and of the most sluggish molecular vibration like the vibrations of the ultra-red, also incapable of affecting the sensorium.

Let us turn again to our study of sapid substances, restricting ourselves in the first case to the investigation of inorganic compounds. A large number, perhaps the greater number, of inorganic compounds are insoluble in water, and are therefore tasteless; we are therefore confined to those which are soluble, and which are chiefly the salts of certain metals. I have endeavoured to obtain information as to the tastes of different substances, by consulting standard works on chemistry, especially the dictionary edited by Watts. In many cases I was unable to obtain any information at all, and in nearly

every case it was insufficient for my purpose. One knows pretty exactly what is meant by a peacock blue, or a salmon colour, but the general descriptions of tastes are absolutely unreliable. Common salt and nitre are both described as saline, sulphate of magnesia and quinine both as bitter. I have been obliged on this account to taste the different substances for myself, and all the observations recorded in this paper are the result of at least two careful experiments with each substance. By the term *salt* I shall indicate a taste like that of common table salt; by *saline*, a taste like that of nitre.

In recent times a remarkable discovery of Newlands has formed a fresh point of departure in connection with the science of chemical physics. His observations led him to formulate a law which he termed the law of octaves. Lothar Meyer, Mendelejeff and Carnelley, in extending his work, have shown that the Periodic Law—as it is now termed—is one of wide application and importance. The nature of the Periodic Law is now so well known, thanks to the many recent publications of Professor Carnelley, that it would be superfluous to do more than merely sketch its main features.

If we arrange the elements in the order of their atomic weights, beginning with that which has the lowest, and passing to that which has the highest, we shall find that there is a periodic recurrence of function or property in the series. The first element is a monad, the second a dyad, the third a tryad, and the fourth a tetrad. Then we find the fifth a triad, the sixth a dyad, and the seventh once more a monad. Then follows a second series of seven elements, showing the same variation in atomicity, and this repeats itself right through the list of elements.

This periodic recurrence is seen not only with atomicity, but with the atomic volume, the fusibility, the electrical and other properties of the elements. There is then a general resemblance in physical properties between the first, eighth, fifteenth, &c.; between the second, ninth, sixteenth, &c., and so on. Mendelejeff has arranged the elements in a very convenient tabular form, see table p. 152, which brings out these and some other important facts.

Those elements which resemble one another, and which we

can pick out, taking every eighth one from that one we start with, form what he calls *a group* (see table). In vertical series are arranged the *groups* of similar elements, and the *series*, each of seven elements, are seen on the horizontal line.

TABLE OF NATURAL CLASSIFICATION OF ELEMENTS. AFTER MENDELEJEFF.

Group	I.	II.	III.	IV.	V.	VI.	VII.	Group VIII.
Series.	Monads.	Dyads.	Triads.	Tetrads.	Triads or Pentads.	Dyads or Hexads.	Monads or Heptads.	
1	H=1	..	..	..	..	..	..	..
2	li=7	be=9	b=11	c=12	n=14	o=16	f=19	..
3	NA=23	Mg=24	AL=27	SI=28	P=30	S=32	CL=35·5	..
4	K=39	ca=40	sc=44	ti=48	v=51	cr=53	mn=55	fe=56 co=57 ni=59
5	CU=63	ZN=65	GA=69	..	AS=75	SE=79	BR=80	..
6	rb=85	sr=87	y=89	zn=90	nb=94	mo=96	..	ru=104 rh=104 po=106
7	AG=108	CD=112	IN=113	SN=118	SB=120	TE=125	I=127	..
8	cs=133	ba=137	la=139	Ce=142	di=147	..	..	..
9	..	..	..	..	ER=166	..	..	..
10	..	..	yb=173	..	ta=182	w=184	..	os=193 ia=193 pt=195
11	AU=197	HG=200	TI=204	PB=207	BI=210	..	..	..
12	..	..	..	th=234	..	u=240	..	..

There is one other point of importance, namely, that the elements of a *group* which are in an *even* series are especially related one to another in their properties. In the table they are represented in small type. So, in like manner, elements belonging to the same group of *odd* series are especially related.

Thus Li, Na, K, Cu, Rb, Ag, Cs, Au have all of them properties in common; but in this group Na, Cu, Ag, Au are most alike, and Li, K, Rb, Cs, also in like manner, are especially related.

Scientific men have long been taught to expect that material substances similar in their ordinary physical properties, will exert a similar influence on organic life. No one has more completely demonstrated this than Drs. Lauder Brunton, Fraser, and Crum Brown, who have shown that not only elementary protoplasm, but even highly differentiated muscular and nervous tissues are affected in a similar way by substances having allied chemical and physical properties.

Every one is aware, too, of the important part played by the component atoms of a molecule, how by replacing an atom of

hydrogen by an atom of potassium one can convert ordinary water into a caustic alkaline substance. Inasmuch as we have a number of salts formed, say, by the union of chlorine with another element, it is a question well worth the discussion, whether salts formed by the union of chlorine with elements of the *same group* have similar tastes. Speaking more generally: do elements belonging to the same group when combined with the same element or group of elements to form salts, similar as they are in physical properties, produce similar tastes?

It was this question which occurred to me, when, baffled in the hope of connecting taste directly with the gross atomic weight of the sapid substance, I had to reconsider the question afresh. This question, I shall hope to show, may be answered in the affirmative.

By far the greater number of sapid substances contain elements found in Groups I., II. and VII., and these I accordingly studied. The salts of Groups I. and II. most easily obtained are the chlorides and sulphates, and of Group VII. I selected the combinations these elements form with sodium and potassium. As it was very necessary to use salts as pure as possible, many were prepared for me with great care by Mr. Harris the chemist, but for the greater number I have to thank my colleague Dr. Nicol, who placed at my disposal a large collection of very pure salts prepared by himself for his own well-known investigation on solubilities.

In the subjoined tables are the results of an investigation of Groups I., II. and VII. We have then to see whether the same taste sensations are produced by the chlorides, &c., of elements having similar physical properties.

GROUP I.

Metal.	Chloride.	Sulphate.
L	Salt	Sal. Bit.
Na	Salt	Sal. Bit.
K	Salt Sal. *Bit.*[1]	Sal. Bit.
Cu	Insoluble	Sal. Bit.
Rb	Salt *Bit.* Sal.	..
Ag	Insoluble	Sal. Bit.
Cs	Salt *Bit.* Sal.	..
Au	Sal. Ast.	..

[1] *Italics* indicate that the sensation is but slightly perceived.

It will be seen that all the soluble chlorides are salt like table salt, although with the higher members we have the taste becoming more saline, a very slight bitter taste also develops. Some salts are astringent, as will be seen in a study of this and of other groups. I do not think this astringency is generally looked upon as a taste at all; depending, as it does, upon the formation of insoluble albuminates, it is purely a local action on the mucous membrane of the tongue, and will not be again discussed.

The sulphates are saline, not salt at all, and in addition they are distinctly bitter. I have not been able to obtain the sulphates of cæsium and rubidium.

Group II.

Metal.	Chloride.	Sulphate.
Be	..	Acid. Sweet. Ast.
Mg	Bit. Sal. Wrm. Pung.	Bit. Sal.
Ca	Bit. Sal. Wrm. Pung.	Insoluble
Zn	Bit. Sal. Wrm. Pung.	Bit. Sal. Ast.
Sr	Bit. Sal. Wrm. Pung.	Insoluble
Cd	Bit. Sal. Wrm. Ast.	Bit. Wrm. Ast.
Ba	Bit. Sal. Pung.	Insoluble
Hg	..	..

In Group II. the chlorides are all bitter salines with, in nearly every case, a warm pungent flavour. They are very disagreeable to taste, and it is long before the flavour leaves the mouth. Beryllium chloride with the sulphate is described as sweet. I have not tasted the chloride; the sulphate is distinctly acid and astringent as well. Beryllium salts then are a well-marked exception to the other salts, giving, as they do, such constant taste sensations. The atomic weight of beryllium was long an open question. I am informed on competent authority that there is now no doubt on this head, and I am at a loss therefore to explain its action as a sapid substance. The soluble sulphates are saline and bitter.

In Group VII. we have tastes produced which are in the main salt. In the case of both the sodium and potassium compounds there is a tendency to the production of the saline and bitter among the higher members of the group, and this is sooner seen in the case of potassium—itself a higher

GROUP VII.

Element.	Sodium Comp.	Potassium Comp.
F	Salt	Salt. *Sal.*
Cl	Salt	Salt. Sal. *Bit.*
Mn	..	..
Br	Salt. *Sal.*	Salt. Sal. *Bit.*
I	Sal. Bit.	Sal. Bit.

member—than in the case of sodium, a lower member of Group I.

From a study of these groups we may learn many important facts. It will be seen that much depends upon the electro-negative group with which the element is combined. Thus sodium combined as a chloride has a different taste from the sulphate of the same element: with the same electro-negative group similar elements give similar tastes, but with a curious and very uniform change as we pass from an element of low to one of high atomic weight. As we shall soon see, a change in physical property may, in like manner, be seen as we pass from a lower to a higher member of a group.

So far then we have reduced taste to a *function* of elements and their compounds, and we see that it obeys laws which are the same for so-called physical properties of these elements. Just as from a knowledge of the components of a compound we can account for its physical properties, so we have to take to pieces the sapid substance, before we get the clue to relationship between its nature as indicated by its chemical and physical properties, and the sensation it produces.

This is much already to learn; but can we go a step further and ask "in what *essential* are elements alike that produce similar tastes?" We turn naturally to the question with which we started, and ask, "do these elements vibrate in any way that is similar?" Rapid advances are being made in the more exact and extended inquiring, how do the ultimate particles of matter move? In a few years, no doubt, the investigations into the ultra-red and ultra-violet spectrum will shed a flood of light on this question, and will enable us to come to some more definite conclusion than at present. Still much is known, and it may not be premature even now to make use of this incomplete knowledge.

No one would expect to find very closely allied spectra when comparing elements even of the same groups, and for this reason. Suppose potassium and lithium to have such a fundamental tone and a series of upper partials each in an exactly similar inharmonic ascending scale, the tones of the heavier—potassium—would all be of lower pitch than the lighter—lithium. An absorption line of potassium in the visible spectrum will correspond in this case to one of lithium in the ultra-violet. Thus all attempts to establish exact mathematical relationships between the wave lengths of similar metals are for the present out of the question. We can but hope to find rough points of similarity, and these are forthcoming. The chlorides of the alkaline earths are nearly related. The lithium and sodium spectra have some points of similarity, especially in their extreme simplicity; also potassium and rubidium, each with their five groups of lines. Then again, chlorides, bromides, and iodides of calcium and barium are nearly related, the lines shifting towards the red end of the spectrum in a way which is nearly proportional with the increase of atomic weight.

Many salts absorb light of different wave lengths, and are in consequence of a definite colour. The colour is an index to this absorption, and to the state of molecular vibration of the salt molecules. In a paper on the colour of chemical compounds, recently published in the philosophical magazine ('Phil. Mag.,' July 1884), Professor Carnelley demonstrated a relationship between salts of metals of the same group in respect to colour. The salts, say, the chlorides, of a group of metals, are much of the same colour, except that, as you pass to the higher members of a group, this changes somewhat, shifting uniformly towards the red end of the spectrum. This is illustrated by the following diagrams taken from his paper, in which only those metals of a group are given, which are in alternate series.

Metal.	Cl.	I.	Metal.	Cr O_4.	As O_4.
Na	White	White	Mg	Lemon Yellow	White
C	White	Cream	Zn	Yellow	White
Ag	White	Light Yellow	Cd	Orange Yellow	White
Au	Yellow White	Golden Yellow	Hg	Red	Yellow

The striking analogy between the above tables and those of taste already alluded to, needs scarcely to be pointed out, it is obvious.

But what does this shifting towards the red end of the spectrum indicate in the case of these coloured salts?

Probably the colourless ones have vibrations of high pitch in the ultra-violet; with a higher atomic weight and slower vibration they gradually absorb the rays of the visible spectrum. The blue rays are first absorbed, and the salt appears yellow; then the green giving orange, and then the yellow and orange rays are also absorbed giving red.

In the case of tellurium chloride, all are absorbed, and the salt is black. Carnelley has therefore demonstrated that the salts of a group have molecular vibrations which are similar, which absorb light and give rise to *colour sensations* which are similar, changing uniformly, however, with increasing atomic weight.

Here then is the whole analogy between taste and sight, and it is most complete in its nature. If a curve be constructed in which the ordinates represent the atomic weights of the positive elements, and the abscissæ a chromatic scale rising from blue, green, &c., to black, we shall obtain a curve indicating that the colours of the compounds are a periodic function of the elements arranged in atomic series. This is best seen in the case of the normal iodides.

Upon the pitch of the vibration depends the colour sensation, as every one would admit: we find in the case of taste a result in every way is the same.

This has been one induction from the study of the inorganic compounds, but a whole field of inquiry had been untouched. As yet no allusion has been made to the carbon compounds, many of which are sapid. One of the most important and interesting facts in connection with the chemistry of the carbon compounds is, that they have what may be termed a *structure*. Thus the formula for common alcohol is C^2H^6O, but one of the hydrogen atoms is found to be especially related to the oxygen, and it is written C^2H^5 (OH). In this case then there is one group (compound radical) C^2H^5 united with another group OH. Now these

groups play the same part that elements do in inorganic compounds, and it is necessary to know this so-called structural formula in order to understand the property of an organic substance. Just as one can replace the hydrogen in water with potassium, and form another substance of different properties, depending upon the nature of the substance added, so, in like manner, one can replace the C^2H^5, or the OH, by other compound radicals, or even elements, and produce a substance whose properties will depend upon the nature of the radical you add. There are then two points for investigation. In the first case, can we find in sapid substances, having, say a sweet or a sour taste, any compound radicals always present, and from whose presence we may say that the taste sensations result? The chemist associates certain properties with the presence of a certain radical in a substance; can we find similar associations with taste sensation?

In the second place, do we find with the spectroscope that the various combinations of a compound radical preserve the character of the vibrations of the radicals, unaltered in any marked degree?

The answer to the first question can be readily given, for it will be sufficient to obtain a list of substances giving a definite taste, and to search for some common or similar radical. If a radical common to all be found, we may safely answer it in the affirmative. There are among the carbon compounds many substances having an acid taste. Here is a list of those most familiar:

Acetic acid .. .. $CH_3\,CO\,.\,OH$

Oxalic acid .. .. $\begin{matrix} CO\,.\,OH \\ | \\ CO\,.\,OH \end{matrix}$

Succinic acid .. .. $\begin{matrix} CH_2\,.\,CO\,.\,OH \\ | \\ CH_2\,.\,CO\,.\,OH \end{matrix}$

Glycolic acid .. .. $\begin{matrix} CH_2\!<\!\begin{matrix} OH \\ CO\,.\,OH \end{matrix} \\ | \\ CO\,.\,OH \end{matrix}$

Glyceric acid .. .. $\begin{matrix} CH\;\;OH \\ | \\ CH_2\,.\,OH \end{matrix}$

Lactic acid .. .. $CH_3\,CH\!<\!\begin{matrix} OH \\ CO\,.\,OH \end{matrix}$

Tartaric acid	$\begin{matrix}CH\,(OH)\,CO\,.\,OH\\ \vert\\ CH\,(OH)\,CO\,.\,OH\end{matrix}$
Benzoic acid	$C_6H_5\,.\,CO\,.\,OH$
Malic acid	$\begin{matrix}CH\,(OH)\,.\,CO\,.\,OH\\ \vert\\ CH_2\,.\,CO\,.\,OH\end{matrix}$
Glyoxylic acid	$\begin{matrix}CHO\\ \vert\\ CO\,.\,OH\end{matrix}$

The above ten examples of substances possessing distinct acid tastes are taken from various classes of organic acids, and they will be seen to have in all cases a common group of elements. This group is a compound radical CO . OH, which is combined in all cases with a different molecule. We have then the same right to impute to this radical the capacity of producing a given taste sensation, as the chemist has of looking upon it as related to certain physical properties of the compounds in which it may be found.

Among sweet substances we find the following:

Glycol	$\begin{matrix}CH_2\,.\,OH\\ \vert\\ CH_2\,.\,OH\end{matrix}$
Glycerine	$\begin{matrix}CH_2\,.\,OH\\ \vert\\ CH\,.\,OH\\ \vert\\ CH_2\,.\,OH\end{matrix}$
Mannite	$C_6\,H_8\,(OH)_6$
Glucose	$C_6\,H_{12}\,O_6$
Inosit	$C_6\,H_{12}\,O_6$
Saccharine	$C_{12}\,H_{22}\,O_{11}$

These are all alcoholic bodies, glycol, glycerine, mannite being, respectively, diatomic, triatomic and hexatomic alcohol. The rational formulæ of the last three and other nearly related substances, such as levulose, maltose, &c., are not at present definitely settled; but it is certain that they contain a radical CH_2OH, found in the first three substances. Thus the formula of glucose, according to Colley, is—

$$CH_2\,.\,OH - CH\,.\,OH - CH\,.\,OH - CH\,.\,OH - CHOH - .\,CHO$$

Monatomic ethyl alcohol is tasteless, and has been alluded to already. It is the simplest alcohol, the polyatomic alcohols referred to having a sweet taste.

We see here again that, with a definite molecular structure, with the presence of certain groups of elements, a definite taste sensation is produced. A chemist examining the rational formula of a substance will predict its properties to an extent which will vary with the extent of his knowledge. We too can predict a property, that of producing particular sensation when applied to the tongue.

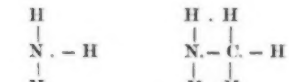
There is a large number of organic compounds having a bitter taste, such as quinine, quassia, strychnia, &c.; but inasmuch as so little is known as to the chemical nature of these bodies, an investigation, if applied to this class of substances, would be of little use.

We have seen that in inorganic compounds the physical, chemical, and taste properties vary with the nature of the elements in the sapid substance; with similar elements similar properties are associated. So too we have seen among the carbon compounds that the physical, chemical, and taste properties vary with the compound radicals present in the sapid substance. The question now to be determined is, whether or not the compound radicals behave as elements, vibrating in a characteristic and definite way, not materially disturbed by altering the combination? This may be determined by the eye, with or without the aid of the spectroscope.

We know that chromic acid has an orange-red colour; combine it with metallic elements to form chromates, and we find that these are coloured, the colour varying very slightly with the metal.

Picric acid is a coloured solid, and it probably owes its colour, at any rate in part, to the presence of a radical (NO_2). Its salts too are coloured, the absorption shifting towards the red end of the spectrum.

In his interesting researches into the spectra of colourless fluids, observed through a considerable length of tube, Dr. W. S. Russell finds that ammonia gives certain well-marked absorption bands. If an atom of hydrogen in the ammonia be replaced by methyl, forming a substance methylamine, the ammonia bands are still seen, shifted however slightly towards the red end of the spectrum.



Ammonia Methylamine.

Replace the hydrogen by the higher group C_2H_5, or ethyl, to form ethylamine, and the shifting continues towards the red.

With a series of alcohols, such as methylic, ethylic, propylic alcohol, we find, as we pass to the more complex, heavier, but similarly constituted molecules, a shifting of equally characteristic bands towards the red end of the spectrum. It is very much (and here we may recall the colour experiments of Professor Carnelley) as if we took an instrument, say a violin, and kept loading its strings. The notes would be heard deeper and deeper in tone, but it would be still recognised as being the tone or quality of the violin.

I have mentioned the above instances, though many others might have been adduced to demonstrate the same fact. We have every reason then to believe that the radical and the radical of the acid and sweet substances are both in constant and characteristic vibration, although this vibration does not effect the rays of the visible spectrum. Not only so, but we know that this vibration will not be altered in character (from the analogy with the ammonia radical in methylamine), being merely shifted to a small extent in pitch, when its combinations are changed. This is, however, the analogy which we have sought.

Both in the case of sight, hearing, taste, and, as I hope afterwards to prove, in the case of smell we have these analogous phenomena. On the side of sensation we have variations in what we may call quality; on the objective side we have matter in vibration—these vibrations vary in character: and it is possible to connect the quality of the sensation with the character of the vibration.

It may be urged that inasmuch as an acid like hydrochloric acid produces much the same taste as oxalic acid, there

is no such definite connection between vibration and sensation. There are, too, some sweet substances having no relationship to an alcohol. We know that one can get the same colour sensation from a solution of either a picrate or a chromate, two substances absolutely dissimilar in chemical properties. The reason is that they both give a group of vibrations in the same part of the solar spectrum. It would be reasonable to expect that instances would be forthcoming of substances of dissimilar nature and chemical composition, with similar tastes. It would seem probable then that there is a scale of vibrations, which may stimulate the taste organs analogous to the visible spectrum. If a substance vibrates in a definite part of this scale, a definite taste will be produced.

One must always remember that the tongue has, like the eye and unlike the ear, no power of analysis. It may be that just as a colour, say orange, may be produced by either a simple vibration, or a combination of vibrations, some higher and some lower in pitch than that of the simple one, so the same taste may, in like manner, be produced by a simple vibration or a compound one made up of simple vibrations of higher or lower pitch.

In conclusion, it will be necessary to state very emphatically that it is *analogy* that this paper proves—analogy in the production of the senses. It would be premature to say definitely how the vibrations of common salt produce irritation of the gustatory nerve. Is it by setting up a sympathetic vibration, when, as in the ear, the sonorous waves set in motion the end-organs of the labyrinth? Upon such a question I have no wish to hazard what could only be an opinion? In the eye, vibrations stimulate the cones. How is this done? It is quite conceivable that this is by setting up sympathetic vibrations, but many suppose it to be a chemical action.

If we ask what is a chemical action, no adequate reply will be forthcoming, though many would hesitatingly affirm that even so-called chemical actions may have a vibratory and mechanical interpretation. When we say that we *understand* the manner in which the end-organs of the ear are stimulated, we mean that we can prove an analogy between the sympathetic vibration of a tuning-fork and of the organ of Corti,

when affected by the same wave motion. It is nothing but an analogy after all.

The production, on the other hand, of end-organ irritation, by light or molecular vibration, has no sufficient definite analogue in the laboratory of the experimenter.

All that I claim to have proved is, that the body is surrounded by vibrating of matter, stimulating its sensitive surface; that in all cases the process of stimulation with the production of a consequent sensation is the same to this extent; that the quality of sensation is dependent upon the character (pitch and complexity) of the vibratory matter.

Just as a certain class of salts of allied chemical and physical properties vibrate in a similar way, and stimulating the eye, produce the same colour sensations; just as certain strings of definite length and consistency vibrate in a similar way, and produce the same sound sensation—so, in like manner, similar sapid compounds (containing similar elements, or the same compound radicals) vibrate in a similar way, and produce the same taste.

ON RECURRENT PALPITATION OF EXTREME RAPIDITY IN PERSONS OTHERWISE APPARENTLY HEALTHY.

BY JOHN S. BRISTOWE, M.D., LL.D., F.R.S.

THE subject to which I wish to direct attention is that of extremely rapid pulsation, occurring for the most part in intermittent paroxysms of variable duration, in hearts structurally and texturally sound, and in persons otherwise healthy.

That hearts may beat with the extreme rapidity with which I have found them to beat, is a fact which, I think, has been largely overlooked, and with which I, at any rate, had no practical acquaintance until within the last two or three years; and yet I feel sure, judging from my recent experience, that the condition which I am about to discuss is of frequent occurrence, and needs only to be looked for intelligently to be recognised in many persons who are regarded as merely nervous and liable to attacks of ordinary palpitation.

So far as I know, the literature of the subject was, until recently, limited to the report in the 'British Medical Journal,' for the year 1866, of three well-marked cases, the first from the pen of the late Dr. Cotton, and the others respectively by Dr. James Edmunds and the late Sir Thomas Watson. Of these cases I need only say, that they almost accurately resembled the most striking and typical of the cases which are incorporated in this paper; that the cardiac pulsations during the paroxysms of palpitation were at the rate of about 240 in the minute; that in the intervals the patients appeared to be well and free from heart disease; and that in Sir Thomas Watson's case (which proved suddenly fatal) the heart was found to be soft and somewhat enlarged, but otherwise healthy.

The first typical case of the disease which I ever fully recognised was one which I saw in consultation with Dr. Wyman, of Putney, in the early part of 1885. The patient was a fairly healthy-looking young married lady, who had evidently been liable for some years to attacks of palpitation, and was free from structural disease of the heart. The attack in which I saw her came on suddenly, without apparent cause, and after a week left her as suddenly as it had arisen. Her pulse varied between 180 and 192 in the minute. A few weeks later she had a recurrence of palpitation, when the cardiac beats were counted at 246. What seemed to me at the time the most remarkable feature of her case was the apparent absence of distress. Had I not known that the patient's heart was beating with extraordinary rapidity it would never have struck me, from watching her and conversing with her, that there was anything the matter with her.

CASE I.—*Paroxysmal Hurry of Heart of Some Years' Duration.*

Mrs. P., a married lady 30 years of age, was attacked about six weeks before I saw her with pleuro-pneumonia of the right side, from which she had apparently recovered completely at the end of about four weeks. A few days before my visit (April 5th) she had taken a drive. This seems to have upset her; for on her return home she was attacked with retching, dyspnœa and nervousness. The retching soon ceased; but the pulse was found to be beating with great rapidity (about 180 in the minute), and this rapid pulsation has continued. She has been kept in bed, complaining of fluttering in the region of the heart, slight dyspnœa and nervousness; but otherwise has appeared fairly well. When I saw her she was sitting up in bed, looking bright and cheerful, but pale and delicate. There was no obvious dyspnœa, although perhaps she breathed a little more quickly than normal, and no cough. The heart was beating regularly at the rate of 192 in the minute. It was not enlarged; and the sounds, which were short and sharp, were unattended with murmur. The lungs were healthy. There was no abdominal affection; the urine was free from albumen, and no anasarca was present. Her tongue was a little furred; and her appetite not very good. There was no goitre or exophthalmos.

The patient had been married for three years, and had no family. She said that formerly she was stout; but that for several years

past she had been, as she now was, thin; and that she had been liable for some years past to attacks which seemed to her like that for which she was now under treatment. They came on suddenly, without cause, and after lasting for a few days subsided suddenly. She had consulted medical men, and had been told that she had heart disease, and had been directed not to exert herself, and especially not to hasten up hill or up-stairs. She had never had rheumatism.

On this occasion the heart continued to act with extreme rapidity for a week, and then suddenly its beats fell to 110 in the minute, and shortly afterwards to 92. I saw her again on the 20th of April. The pulse was beating at the rate of 92 in the minute, and quite regularly. There was no cardiac murmur, and she appeared to be, and expressed herself as feeling, perfectly well.

About a month or six weeks later she had another similar attack, in the course of which the pulse reached 246 in the minute. It was unattended with dyspnœa, and there was no rise of temperature.

My second case was admitted into St. Thomas's in July of the same year. It was that of a man between 30 and 40 years of age who had suffered from heart disease ever since an attack of acute rheumatism eight years previously. He had obstructive and regurgitant aortic and regurgitant mitral disease, with hypertrophy and dilatation of the heart. There was reason also to believe that the arch of his aorta was dilated. He presented all the usual signs and symptoms of abundant aortic regurgitation. It appeared that for several months before admission he had been liable to attacks of palpitation; and during the three months he remained under my care he had many such attacks. They came on suddenly at irregular intervals, night or day, without obvious cause, continued for periods which varied between half an hour and thirty-six hours or more, and subsided suddenly. While in his attacks, he complained of feeling ill, faint and weary; and his pulse ranged from 160 to 200 in the minute. In the intervals it varied from 80 to 100. He came under my care for the second time early in 1886, and remained in hospital for a period of four months. For the first two months he suffered, precisely as he had done previously, from recurrent attacks of palpitation, after which I treated him for the first time

systematically with digitalis and iron; and with the best results, for the attacks at once diminished, and during the last six weeks of his residence in hospital, he remained entirely free. He died shortly afterwards; but I have not been able to obtain any information as to the circumstances of his death.

CASE II.—*Double Aortic and Mitral Disease, and probably Aortic Aneurism; Ascites; Paroxysmal Hurry of Heart; Death.*

William N., a dealer, 34 years of age, first came under my care on the 30th of July, 1885.

At the age of twenty-six he had a severe attack of rheumatic fever, and has never felt well since. A few months ago he began to suffer from palpitation, short breath and cough, which often came on in severe paroxysms, and finally prevented him from doing his work.

He was an exceedingly pallid man, complaining of the symptoms above enumerated. The præcordial dulness was extensive. The heart was much enlarged; its apex beating in the 6th interspace, about three inches below, and an inch and a half to the left of the nipple. There was marked pulsation over the whole of this area, and also in the inner part of the right second and third intercostal spaces. The action of the heart was regular, and attended with a musical systolic murmur at the apex, and a well-marked double murmur at the base. There was some crepitation at the lower part of both lungs. The cough was attended with a good deal of frothy expectoration. The urine contained a trace of albumen.

The patient remained in the hospital until the 31st of October, presenting in a greater or less degree the symptoms above described, and suffering also from great distress and weariness, and not unfrequently from pain extending down the left arm. The most interesting phenomenon in his case, however, consisted in the frequent supervention of attacks of palpitation. These came on suddenly, without definite cause, sometimes in the night, sometimes in the day, and ended suddenly, also without definite cause. They lasted from three-quarters of an hour to 36 hours or more at a time; and while present the rate of the pulse (which for the most part continued regular) varied from 160 to 200 in the minute. Most commonly it was about 180. The attacks came on irregularly, sometimes every day, occasionally even twice a day, and often at intervals of several days. During them the patient felt ill, faint and weary, but did not suffer from cardiac pain. He complained of

dyspnœa, but the respirations were for the most part only about 32. He was liable to slight rises of temperature; but there was no obvious relation between them and the palpitation. In the intervals between his attacks the pulse varied from 80 to 100, was generally regular, and presented a well-marked aortic regurgitant character. Various remedies were given with the object of arresting the palpitation. But latterly nitro-glycerine alone was employed, in doses varying from $\frac{1}{100}$ to $\frac{1}{25}$ grain. This was thought to relieve him at times. But I am doubtful whether it had any real influence over the action of the heart.

On the 12th of February, 1886, he was again admitted under my care. He had suffered during the interval from frequent attacks of palpitation, and latterly his belly and legs had become swollen, and he had had severe attacks of epistaxis. His abdomen was tapped on the 4th of March, when 14 pints of serum were removed; on the 27th, when $12\frac{3}{4}$ pints were taken away; and lastly on the 18th of April, when the amount withdrawn was 13 pints. After the tappings the liver was found to be considerably enlarged. The removal of the ascitic fluid relieved the patient's breath, and rendered him generally more comfortable, but had no material influence over the attacks of palpitation.

These continued, in fact, without change of character up to the 13th of April; they were just as severe, just as frequent, and just as irregular in their occurrence and duration as they had been during his former stay in the hospital. On the 13th of April, with the object partly of benefiting the dropsy, partly of influencing the attacks of palpitation, a mixture, containing ten drops of tincture of digitalis, five grains of tartrate of iron and calumba, was ordered to be taken every six hours. This was five days before the last of the three tappings above mentioned. During the next week or two there were two or three short attacks of rapid action of the heart, and then they ceased altogether; so that during the remainder of his stay in the hospital, a period of about six weeks, he continued quite free from them. Moreover, although for a short time the ascitic fluid seemed to be re-accumulating, it soon ceased to collect; and when he left the hospital there was little or none remaining. Nevertheless, for some two or three weeks after the last tapping, although not suffering from palpitation, he was extremely ill, very weak, very drowsy, inclined to ramble at times, and had several severe attacks of epistaxis. Then he improved somewhat, and though weak and ill when he left the hospital, was, on the whole, a good deal better than when he was admitted. During both his residences in the hospital his urine contained more or less albumen, and his temperature varied from

97 to 101. In the intervals between the attacks of palpitation his pulse ranged from 80 to 110, and was apt to be irregular. And after the last tapping his respirations were, on the whole, more rapid than they had been previously, and often rose to 40 or even 50 in the minute.

He left the hospital on the 7th of June, apparently much better than he was when admitted, but still very ill. He died at home three or four weeks later.

My next case was that of a lady, 49 years of age, who for several years had had a largish goitre; but, so far as could be ascertained, had not suffered from palpitation, or other symptoms of Graves's disease. When I saw her she had been complaining for two or three weeks of cough, dyspnœa, and great rapidity of heart's action. It had been ascertained on several occasions that the beats were about 200 in the minute. At the time of my visit she was in bed, complaining of a frequent dry cough and palpitation; but in other respects seemed to be in fair health. The heart (the apex of which was somewhat displaced to the left) was beating at the rate of 180 in the minute, and somewhat irregularly. Its sounds were free from murmur, and no evidence whatever of disease either in the thorax or in the abdomen was discovered. I saw her again three days later. She was then very restless and ill, and her cardiac pulse, which had been 240 the night before, was now about 220. There was still no discoverable visceral disease. The next day her urine was found for the first time to contain a good deal of albumen. She died the following night. I am inclined to think that the goitre in this case was a mere accident of the case, and did not imply the presence of Graves's disease, and to suspect that the attack in which the patient died was only the last of an unrecorded series of such attacks.

CASE III.—*Goitre—Rapid Action of Heart—Restlessness—Albuminuria—Death.*

Mrs. P., aged 49, a patient of Dr. Wyman's, of Putney, has, on the whole, had good health; but for several years has had a largish goitre, which is said latterly to have caused some difficulty of swallowing. She has been treated with iodide of potassium,

and, it is believed, with benefit. There has never been an exophthalmos, or stridor, or, so far as is known, attacks of palpitation.

Her present illness began two or three weeks before I was asked to see her, on the 16th of February, 1886; and has been characterised by cough without expectoration, shortness of breath, and great rapidity, with some irregularity, of heart's action. Her illness, however, has not prevented her from performing her accustomed household duties; and it was only yesterday that, by Dr. Wyman's orders, she took to bed. Her pulse has been several times counted, and found to be about 200 in the minute. Her appetite has been poor, but she has slept well, and her urine has been free from albumen.

She was a healthy-looking woman; and, as she lay in bed, seemed free from dyspnœa and all other kinds of distress. The goitre was rather large. There was no lividity or anasarca. Her tongue was clean. She complained of cough, but it was not severe, and there was no expectoration. No evidences of disease were discovered in the lungs. The præcordial dulness was rather large, and the heart apex was thrown a little outwards, but there was no undue prominence. The pulsation was somewhat diffused. The heart was beating slightly irregularly, at the rate of 180 in the minute. The sounds were short and sharp, but free from murmur. No pericardial friction; pulse weak, somewhat irregular. No evidence of abdominal or renal mischief.

I saw her again three days later (the 19th). She was then very ill. It appeared that she had been getting worse, and that since yesterday she had been extremely restless, rarely sleeping, and then only for a few minutes at a time, and constantly rambling. There was no sickness; but she had taken little food, and the bowels had been somewhat confined. Her evacuations had been passed consciously. The pulse last night had been 240. The urine had been free from albumen; the temperature a little above the normal. She was now exceedingly restless, tossing her limbs about, and shifting her position constantly. She was rational, but her articulation was very indistinct. Skin dry, but not hot; tongue thickly coated. Occasional cough, but no marked difficulty or acceleration of breathing. Chest resonant, breath-sounds healthy. Præcordial region as before. No pericardial or cardiac murmurs. Action of heart somewhat irregular, and varying from 200 to 220 in the minute. No paralysis, no affection of pupils, no fits—nothing in fact to point to cerebral mischief. Goitre unchanged.

I heard the next day that the patient was still extremely restless; that her pulse continued about 220; and that she seemed

sinking. The urine now was found to contain a considerable quantity of albumen. She died either that night or the next morning early.

The case of Miss J., which follows, is one of remarkable interest. She was a highly intelligent and active-minded woman, and for three or four years had occupied a post which not only involved continuous official labour and much responsibility, but required her two or three times a year to make a tour of inspection embracing some of the larger provincial towns. Her attacks of palpitation began about the year 1870, when she underwent a long spell of hard work and anxiety, which was followed by a long period of sleeplessness. The first attack came on quite suddenly, and after half an hour ceased suddenly. Subsequently she had many such seizures, coming on at variable intervals, often of many months, and usually without apparent cause. They increased latterly, but rather in duration than in frequency. The attack immediately previous to the one in which I attended her, came on towards the end of 1885, and lasted for six weeks, during the whole of which time her cardiac pulse was regular, and varied from 200 to 260 in the minute; and she continued to do her official work, and generally to act as though she were a healthy woman. Her next attack came on on the 10th or 11th of April, 1886, and ten days later I saw her for the first time. She was then suffering from a troublesome cough, and complaining (as was usual during her attacks) of fidgetiness, and of feeling in a hurry; but otherwise she seemed well, and was performing her official duties as usual. Her cardiac pulsations had ranged between 200 and 250 in the minute, and at the time of my visit were 216. There was no obvious enlargement of the heart; its sounds were free from murmur, and no evidence of disease was discovered either in the other thoracic or in the abdominal organs. At the end of five weeks the pulse suddenly fell to the normal, and for five days continued normal. On the 10th of May, without warning, the palpitation recurred; and, while still suffering from it, she determined to go on one of her periodical tours of inspection. She was feeling very poorly

when she left London on the 1st of June; on the 4th of June, for the first time in her life, her legs became swollen; on the 9th, she reached Liverpool, and feeling very ill, telegraphed for her brother-in-law to fetch her. Nevertheless on that day she spent six hours in inspection. It only remains to say that from this date she suffered from anasarca, pulmonary apoplexy and pleurisy, that the rapid action of her heart continued, and that finally she died with the usual symptoms of cardiac inadequacy on the 10th of July.

It is remarkable that, excepting during her last attack of palpitation, which was unusually prolonged and doubtless aggravated by persistence in the performance of laborious work, Miss J. suffered little while her heart was beating with extraordinary rapidity, and was able to take long walks, and to perform systematic and laborious mental and bodily work. She usually complained of discomfort and worry when the palpitation came on, but soon apparently became reconciled to it, only feeling somewhat irritable and restless. It is remarkable, too, that there was no clear evidence even of cardiac hypertrophy or dilatation.

CASE IV.—*Paroxysmal Hurry of Heart and Restlessness of Fifteen Years' Duration—Death with Symptoms of Cardiac Obstruction.*

I first saw Miss J., aged 40, with Dr. Floyer on the 21st of April, 1886. She had then been suffering for ten days, and was still suffering, from an attack of palpitation.

Her history was as follows:—She had had good health down to the time of the Franco-Prussian war, when, owing to circumstances on which it is needless to enter, she was for some time overworked and had much responsibility thrown upon her. Following on this she suffered for some months from sleeplessness, and had to take chloral. It was about this time that she had her first attack of palpitation. It came on suddenly and left her suddenly, lasting for about half an hour. This attack was followed by others, which seized her at irregular but long intervals, often of many months. These have increased upon her of late, but rather in duration than in frequency. They have come on without warning, and for the most part without obvious cause; but she thinks they have sometimes been induced by dancing and excitment. The last attack occurred in the latter part of last year, and continued

day and night for about six weeks. Like all its predecessors, it began and ended suddenly. The palpitations have always been a cause of distress to her; but have never prevented her from taking exercise or performing whatever duties she has had to perform. For the last three or four years she has been a Government inspector of needlework, and her employment has obliged her to spend some hours daily in official work at Westminster, and periodically to visit various towns in England for the purpose of inspecting the needlework at certain schools; and she has performed these duties thoroughly, even while the attacks of palpitation have been on her, and apparently with injury. What has been the rate of the heart's contractions during the numerous attacks she has experienced I cannot say; but in the last one it certainly varied between 200 and 260 in the minute. The higher number was more than once counted by Dr. Sharkey, who was seeing her at that time. She says that she always feels much distressed when an attack first comes on; but that afterwards she gets accustomed to it, and the distress diminishes; and that she cannot lie on her back or left side on account of the unpleasant throbbing of her heart; but otherwise is unconscious of its rapid action. Her main complaint is that when the palpitation is on her she always feels irritable, fidgety, and in a hurry as if (to use her own expression) she was impelled "to do three days' work in one." She has not been troubled with shortness of breath. She has never had rheumatism, chorea, enlarged thyroid or exophthalmos. She has not been hysterical, and the catamenia have been regular.

Her present attack, as has been stated, came on suddenly ten days ago, and has continued ever since, her pulse ranging the whole time between 200 and 250 in the minute. She has had an irritating cough upon her for some days, which seems to have added to her discomfort; and consequently for a day or two she has remained at home.

She was a healthy-looking woman, apparently not suffering much distress; and indeed no one to look at her or to converse with her would have thought there was anything the matter with her. There was no lividity or puffiness of face, no distress of breathing. Now and then, however, she had a rather violent paroxysm of coughing, attended with little or no expectoration. On examining the chest, the heart was found to be beating at the rate of 216 in the minute, and regularly. Its sounds were short and sharp, and quite free from murmur. The apex of the organ was not displaced; and the præcordial dulness was not enlarged; but the pulsation of the heart was diffused. The lungs were resonant and

the respiratory sounds were normal. There was no sign of abdominal affection. The urine was free from albumen. She was eating and sleeping fairly well. I may mention here that she was a singularly bright, intelligent and well-informed woman; and not only took great pleasure in her official duties, but was generally well-informed, and could talk sensibly and pleasantly, and with piquancy, on many subjects.

The next time I saw my patient was on the 3rd of May. I called on her at her office late in the afternoon. She had been performing her daily duties ever since I last saw her; and now, having been at her post fully employed from an early hour in the morning, was preparing to leave. She looked well, conversed freely and without difficulty, and said that she felt well but for the feeling of unrest, and the impulse to hurry, which were always present when her heart was acting rapidly. The heart was beating at the rate of 208 in the minute; and had been going on thus without intermission for twenty-two days. She still had a little cough.

I did not see her again until the 30th of May. I learnt then that she had awoke on the morning of the 5th of May with her pulse at 120, and feeling quite well; that in the course of the day the pulse had fallen to its normal rate, varying between 70 and 80; and that for five days she continued absolutely healthy. This subsidence of the rapid cardiac action was not due to any obvious cause. She had been working hard and sleeping badly down to the very moment of its occurrence. On the 10th, without manifest reason, the morbid action of the heart recommenced suddenly, and it has continued ever since.

She was not feeling well at the time of my visit; she had been sleeping badly, her appetite had been poor, she had occasionally been retching in the morning, and she had felt weak; moreover she had been suffering from cough. But she had been doing her work thoroughly, and contemplated starting on a visit of inspection to Durham, Liverpool and some other towns. Her heart at this time was beating regularly, and at the rate of 232 in the minute. There was no murmur; the sounds were quite distinct; but there was scarcely any interval between the first and second. The pulse was weak, and could not be counted at the wrist. Her breathing was quick, but I was unable to determine its rate because it always underwent unintentional modification when I tried to count it. But she did not complain of dyspnœa, and spoke as usual freely and without difficulty. There was no lividity or anasarca. The lungs were normal.

On the 1st of June she started alone for the north, as she had

intended, and visited amongst other towns Durham, and lastly Liverpool. She felt ill when she left London, and continued feeling ill during the whole of the time she was away. She suffered from a noisy, distressing and paroxysmal cough. On the 4th of June for the first time in her life her legs became œdematous, and the œdema increased during the rest of her journey. On the 9th she spent six hours in inspecting a school at Liverpool; but she did it with difficulty, and in the evening was brought up to town by her brother-in-law, who had been sent for to her from London.

I paid her a visit on the 10th. She was then extremely ill. Her breath was short; her cough (unattended with expectoration) was very troublesome and violent; she complained of much pain in the præcordial and epigastric regions and across the lower part of the back. Her tongue was clean, but she had loathing of food and frequent sickness; no affection of fauces could be discovered; the lungs were resonant, and the breath-sounds normal; the heart was beating regularly and without murmur, at the rate of 208 in the minute; there was tenderness below the ribs in the hepatic region, and apparently slight enlargement of the liver; and the urine was scanty; her legs were œdematous, and she perspired profusely.

Afterwards she passed a very restless night, suffering much from cough and dyspnœa, and from præcordial and epigastric pain. And when I saw her on the afternoon of the 11th she was much worse than I had yet seen her. She was perfectly sensible; but the præcordial pain, and distress were so great, her breathing was so difficult, each recurring paroxysm of cough was attended with so much aggravation of her sufferings, and she was so collapsed, that her life was believed to be in imminent danger. Her pulse was regular, and 240. There were no abnormal cardiac or respiratory sounds. A few whiffs of chloroform gave great relief to her cough; and in the belief that her sufferings were largely due to over-distension of the right side of the heart and to congestion of the liver, twenty leeches were applied to the præcordial region. These bled freely, and gave her great and immediate relief.

When I visited her on the 12th, I found that she had had a fairly comfortable night, and that she continued much easier. But she was very weak, had occasional attacks of dyspnœa, her cough, though reduced in severity, continued, and her legs were still anasarcous. She did not complain of palpitation, but her heart was beating 240 in the minute. Her respirations were 40. The urine, for the first time, was found to contain a little albumen, but no casts were discovered. The chloroform still relieved her cough.

During the next day or two there was no very great change in her condition on the whole; she had fairly good nights, owing probably to the subcutaneous injection of morphia, her cough was better, but her appetite was very poor, and at times she went off into an alarming state of collapse.

On the afternoon of the 14th she was attacked with agonising pain in the region of the heart—so severe that she was constantly clutching at the left breast with her hand, rocking herself to and fro, and crying out and groaning. She was very pale and ghastly-looking, but not distinctly livid. The pain was continuous, was much like that she had suffered from previously, and did not appear to be true angina pectoris. The inhalation of nitrite of amyl was had recourse to, however, and with some degree of benefit. Chloroform was subsequently employed. And between them, these remedies, and the application of dry mustard to the chest, gave considerable relief. During this time her pulse was 208, and her respirations 40; and for the first time I detected a little crepitation at the base of the left lung. Her temperature was 100·4.

About 7 P.M. Dr. Floyer gave her 30 grains of bromide of potassium, and at 7.40 injected one-eighth grain of morphia, after which she had some sleep. About 9.30 she awoke, and felt what she termed a "lilt" in the region of the heart, or as if "one of its cogs was broken," she became at the same time more faint than she had ever felt before, and thought she was dying. Her sister came to her at once, and found her almost insensible and perfectly quiet, with cold extremities and imperceptible pulse. A minute or two later her brother-in-law arrived. She had already rallied somewhat; but she was still much prostrated, her hands were cold and clammy, and her surface generally perspiring profusely. Her pulse had fallen to 136. She soon recovered from her collapse, and subsequently passed a comfortable night.

On the next day, the 15th, she was much better than she had been for some time. She had little or no distress of any kind; her appetite had improved; her cough had almost subsided; she had no pain; she looked bright; her pulse (which could be easily counted at the wrist) varied between 128 and 132; the temperature was 99; and the urine (which had a specific gravity of 1008) was free from albumen. I thought, also, that the dropsy of the legs had diminished.

From this time down to the evening of the 23rd, the improvement seemed to be maintained, if not to progress. She slept fairly well, and for the first time since her journey to the north was able to lie down; her appetite improved; her pulse varied from 112 to

128; and she "felt at peace." Also the dropsy seemed to be diminishing. But her cough became a little more troublesome; there was some crepitation at both bases, and her temperature was febrile, on one occasion rising to 102. On the 23rd she had seemed as well as she had been during the previous few days. In the evening she took 20 grains of chloral, and slept comfortably for an hour or two. Then she woke up suddenly with a severe stitch in the right side of the chest; shortly afterwards had a very severe and painful attack of coughing; and while it was in progress felt a sudden throb in the region of the heart. The pulse at once jumped to 180 or 185 in the minute; and all the old sense of nausea, distress, and hurry returned. When I saw her on the afternoon of the 24th, she looked ill; her tongue was coated, and her appetite gone; she still had a stitch in the right side, and was breathing 40 in the minute; and her pulse varied between 200 and 208. There was no dulness on percussion over the lungs, but loud pleural friction was audible over the whole front of the right side. Her temperature was 100. Ten leeches were applied to the affected side, and much relief to her symptoms ensued.

For the next fortnight there was no material change in her condition, excepting that during the whole time her cough, which was troublesome, was attended with hæmoptysis. The blood was mixed with mucus, was variable in quantity, but never abundant, and generally brownish or rusty-coloured. It was evidently the hæmoptysis of pulmonary apoplexy, to which the pleurisy also no doubt was due. The pain and friction in her right side subsided, and the sickness and loathing of food diminished. But her pulse continued at the rate of 208 in the minute; her breathing was somewhat rapid; and it was thought there was a little fluid in the left pleural cavity; she complained at times of pains in the hepatic region; the swelling of the legs continued, although it was thought that it was gradually diminishing; and her temperature remained elevated by a degree or two. It may be added, that the action of the heart continued to be regular; that no cardiac murmur was ever detected; that there was no clear evidence that the organ was enlarged; and that the veins in the neck were not dilated.

I was sent for on the morning of July 10th, and reached her at 1.30 P.M. I learnt that she had been attacked during the course of the previous night with extreme dyspnœa, which had continued. When I saw her she was moribund. She was sensible, but disinclined to answer questions; she was breathing quickly with rattles in her throat, moaning and frequently calling out for relief; her face was extremely pale, her extremities were cold and her pulse at

the wrist could not be felt; but her heart was acting regularly, and still about 200 in the minute. She died at 3 P.M.

No autopsy was made.

My fifth case possibly does not belong to the same category as those which precede; at any rate irregularity of cardiac action was as marked a feature as frequency of action, and the palpitation (so far as I know) was persistent. The patient was a spare, healthy-looking, elderly man, accustomed to field sports. He had had, he said, "a sunstroke" a year previously, and had been liable to headaches ever since. Five or six months before I saw him, it was discovered accidentally that there was something wrong with his heart. I saw him on two occasions, and on both found his heart beating irregularly at the rate of between 144 and 148 in the minute. He was not nervous at the time, had no cardiac discomfort, and indeed (excepting for the fear that he had heart disease, as he had been told he had) felt and looked quite healthy. There was no evidence of enlargement of the heart and no murmur. And he assured me that within the last few weeks he had run three miles in twenty minutes without distress.

CASE V.—*Sunstroke (?)—Headache—Rapid Action of Heart.*

Mr. S., a spare, healthy-looking country gentleman, aged 65, accustomed to shooting, riding, and out-of-door exercises, called upon me on the 21st of June, 1886. He has had excellent health down to a year ago, when on a very hot day, being on the seashore in command of a Volunteer Artillery Corps, he had what was called a sunstroke. What the symptoms were, beyond severe pain at the top and back of the head, I do not know. He does not appear, however, to have had a fit. This pain continued for three weeks, and since that time he has been liable to frequent recurrences of it. For some time past it has been a dull aching all round the head, but mainly on the left side, where it becomes acute when he sneezes or coughs. It is generally absent at night and in the early morning; but it comes on after luncheon, and increases until bed-time. He sleeps well. He suffers slightly from indigestion, and his appetite is not very good. For two months in the early part of the year he was confined to bed with some inflammation of the right big-toe. This was, so far as I can

make out, certainly not gout. He has suffered from occasional rheumatic (?) pains for years. It is stated that the medical man who attended him when his foot was bad, detected "slight valvular disease of the heart."

At the time of his visit he was feeling very fairly well; and I should have regarded him as being healthy had not I found his heart beating at the rate of 144 in the minute, and somewhat irregularly. He did not appear to be, and said he was not, nervous; and, indeed, his cardiac pulsations underwent no change during the whole of our interview. He was not himself conscious that there was anything unusual in the action of his heart; had no uneasy sensation in the præcordial region, and had no shortness of breath. He assured me, in fact, that within the last few weeks he had run three miles in twenty minutes, without the least distress. There was no evidence of enlargement of the heart, and no trace of murmur. All his other organs appeared to be perfectly healthy. His urine was free from sugar and albumen. I ordered him a mixture containing digitalis, iron, and ammonia, and some *cannabis Indica*, in the form of a pill.

On the 30th I saw him once more. There was no material change. His heart was beating at the rate of 148, and apparently quite regularly; but the beats at the wrist were unequal in force, and occasionally imperceptible, so that the radial pulse felt irregular. No cardiac murmur, no dyspnœa, no anasarca. He complained only of headache at times, lassitude, and impairment of appetite.

Mr. C., my next patient, called on me about a week later than the last. He had consulted me in 1879, at which time his heart was acting with much irregularity and frequency. Although I could discover no definite sign of heart disease, and he complained little of distress, I confess I took an unfavourable view of his case. He got well, however, while taking iron and digitalis. In 1883 I saw him again, when he was suffering from a similar attack; and he informed me that he had had such attacks occasionally ever since I had seen him. When he came to me in June, 1886, he looked, and on the whole felt, well; but his heart was beating with slight irregularity at the rate of 168 in the minute; and he furnished me with a written statement of the number and duration of attacks of palpitation he had had during the previous eighteen months. From that time until his death

in June, 1887, the action of his heart continued irregular and rapid; and although never, so far as I know, attaining 200 beats in the minute, frequently I believe presented a rate of 300 beats per minute for a few seconds at a time. He continued apparently well (excepting for the rapid action of his heart) until about a month before his death, when the ordinary symptoms of obstructive heart disease came on.

CASE VI.—*Paroxysmal Hurry and Irregular Action of Heart of Seven Years' Standing—Death.*

Mr. C., a short, healthy-looking, commercial gentleman, about 38 years of age, called on me on the morning of the 28th of June, 1886. He told me that he had been attacked suddenly on the 25th with violent palpitation of the heart, which had continued without intermission ever since. It came on without definite cause. He has felt poorly and disinclined for work, and indeed has taken a holiday from business, but has not felt seriously ill. On presenting himself before me, he looked, and spoke, and acted exactly as a healthy man might do; there was no obvious distress, no hurry of respiration, no congestion or lividity of face, and no anasarca. But his pulse was 168 in the minute, and slightly irregular. The præcordial distress was not more extensive than normal; and the apex of the heart impinged in the usual place. The sounds were short and sharp, and quite free from murmur. There was no further evidence of disease anywhere.

About the year 1875 he had syphilis, followed by secondary symptoms, which have never recurred. In Sept. 1879, he was brought to me for consultation, by Dr. Johnstone English. Two months previously he had begun to complain of uneasiness at the pit of the stomach, supposed to be due to indigestion; this had continued, and it was discovered that his heart was acting with great irregularity. At the time of his visit to me he was pale, he complained of slight shortness of breath, his urine contained a little albumen, and his heart was acting very rapidly and with much irregularity. But I could discover nothing else. He had no cough, no anasarca, the lungs were free from congestion, and the heart was apparently of normal size, and its sounds were unattended with murmur.

I was much puzzled about the case, for there was nothing whatever in the history (excepting the previous attack of syphilis) to suggest a cause for the symptoms, and there was nothing to prove the presence of organic heart disease. It was agreed, however, that

he should abstain from business (which he had been attending to hitherto) for a month, and should take iron and digitalis. He got apparently quite well in a short time.

In November, 1883, he called upon me again. He told me he had had good health, excepting that at irregular intervals he had had attacks of palpitation, which were always relieved by the medicine which Dr. English and myself had prescribed for him. He was now suffering from an attack. He complained of a little shortness of breath in going up-stairs, and that he was conscious of the beating of his heart. But otherwise he felt well, and he looked well. There was no anasarca. The heart was beating very quickly and irregularly, and no murmur was to be detected. No definite cause could be assigned for the attack, unless it was that he had been sitting up late. He was a bachelor, and I suspected his habits were irregular.

When he came to me in the present year, he told me that he had continued liable to attacks of palpitation; and he furnished me with the dates and durations of his attacks since the beginning of 1885, as follows:—March 30th, 36 hours; April 13th, 33 hours; May 19th, 18 hours; July 29th, 57 hours; August 2nd, 4 hours; October 22nd, 46 hours; Nov. 25th, 44 hours; Jan. 6th, 30 hours; Jan. 31st, 22 hours; Feb. 24th, 12 hours. He could not always assign a cause for the attacks, but occasionally he thought they followed a little over-indulgence. They seemed always to be benefited by recourse to the iron and digitalis.

He called upon me again on the 1st of July. He had not been feeling well; but had slept well, and looked well. His pulse was beating between 120 and 130 in the minute, and was very irregular. On the 7th he again paid me a visit. He was much better; and ate, slept, and looked well. But he still complained of a little cardiac discomfort. The pulse was irregular and beat 80 times in the minute; but if it had been acting regularly at its generally prevalent rate, the beats per minute would have been 100. His normal rate of pulse, he told me, was about 60. He had been taking his old prescription. It was decided that he should take a few weeks' holiday on the Continent.

Termination of the Case.—The above case was written out for publication, together with several of the other cases, very nearly 12 months ago. The patient has died since, and I here add a brief statement of his subsequent history.

After I saw him in July, he went to Zermatt for a month. He did not climb, but habitually walked three or four miles a day; and returned benefited in general health by his trip, but still with a rapid irregular pulse.

He called upon me in the latter part of February of the present year, complaining of what he called indigestion. This seemed to consist mainly in an aching pain across the upper part of the belly induced mainly by taking exercise. The heart was beating irregularly, 160 in the minute; and I thought the abdominal pain was due to hepatic congestion. On the 2nd of April I saw him again. He was still complaining of abdominal pain; but in other respects felt, and he looked, well. I examined his heart very carefully on this occasion. Its action was very irregular, presenting two distinct alternating rhythms, each lasting for two or three seconds. In the one the heart was beating at the rate of 80 to 100 in the minute; in the other, at the rate of at least 240. There was diffused cardiac pulsation, but the præcordial dulness was not enlarged, the apex beat in its normal situation; there was no murmur, and the second sound at the left base was not accentuated. On May 4th he paid me another visit. He still had the epigastric pain, and for two days had been suffering from diarrhœa. I again examined his heart carefully. It was beating as before. But on several occasions during the brief periods in which it was beating most rapidly, I counted quite distinctly at least ten beats in two seconds; so that quite certainly, although the heart was not accomplishing more than about 160 beats in the minute, these were frequently at the rate of 300 per minute. After this he went to the Isle of Wight for a few days; but he became very ill there, and returned to London in the middle of May, suffering from great difficulty of breathing, general anasarca, and pain and tenderness in the region of the liver. He was livid, and his urine presented a trace of albumen. Dr. Wilbe was called in to attend him, and I met him in consultation. It is sufficient to say that, although the pulse on the whole became more slow and regular, the usual symptoms of obstructive cardiac disease continued; pleurisy with effusion on the right side, for which he had to be tapped on two occasions, supervened; and he died on the 9th of June. No post-mortem examination was permitted.

My seventh case is in many respects the most interesting of the series. The patient was a young man, whose illness apparently dated from a strain twelve years before. There is some reason to believe that, ever since that time, he had had occasional attacks like that for which he came under my care. This had already lasted three months when I saw him; he had only been laid up, however, for six weeks, and latterly he had had some œdema about the ankles. During

the earlier part of his stay in the hospital, his pulse varied in rapidity, often rising to 250 or 260 in the minute, and often presenting considerable irregularity of action, the beats at such times being apt to present sudden alternations of rate between 240 or 250 and half that number. At this time he complained of sickness, aching across the upper part of the belly, and attacks of faintness, associated with lividity and dyspnœa; moreover, the slight anasarca continued, a little albumen appeared in the urine, and he spat a little blood. After a short time he was treated with iron and digitalis, and much benefit resulted; for although his cardiac pulse continued variable, at times quick and at times irregular, it became comparatively slow after a few days, and then fell to 50 or 60 in the minute. On one occasion it sank to 34. With this change the other symptoms of cardiac disease (anasarca, hæmoptysis, albuminuria, &c.) all disappeared, and he left the hospital apparently well at the end of a month. He thenceforth visited me at intervals as an out-patient. For a month he remained at home, and then resumed his occupation. While thus attending, he reported himself as being on the whole fairly well, but liable to attacks of faintness and palpitation. At the time of his visits to me his heart was often found beating at the rate of only 70 or 80 in the minute; but it was liable to be irregular in its action, and its rate would often rise suddenly to 250 or 260. On one occasion I counted the cardiac beats to be 308 in the minute. On the whole the patient seemed to be going on well, but twelve weeks after he first came under my care, and at a time when he seemed to be in the enjoyment of good health, he died quite suddenly. It was noted during life that the heart was enlarged, and that always when it was acting at a healthy rate there was a systolic murmur at the apex. It was believed, however, that the valves were really healthy. At the autopsy the heart was found to be somewhat large and dilated; but the muscular tissue and the valves were healthy, and the ventricles were empty.

CASE VII.—*Rapid Action of Heart—Sudden Death—Autopsy.*

Arthur W., a draper's assistant, 19 years of age, was admitted into St. Thomas's, under my care, on the 4th of December, 1886.

He has had most of the ordinary diseases of childhood, but never any serious illness. When 8 years of age he took part in a paper-chase, but after running across two or three fields, had to stop suddenly on account of a severe pain in the region of the heart. He leant against a tree, and was speechless for some minutes; but was able to walk home. After that, running was prohibited for a time. He seems to have been liable to palpitation on exertion, especially on running up-stairs, ever since; but has otherwise had excellent health up to three months ago, from which time he dates his present illness.

He was then attacked with general aching pains and sense of poorliness, some cough, and shortness of breath. The symptoms persisted, but nevertheless he continued to follow his employment for six weeks. At the end of this time, his abdomen was swollen, and his legs were œdematous, and his palpitation and dyspnœa had increased. Latterly, too, he has complained of aching across the upper part of the belly, and of vomiting. He has had syphilis.

The patient was a tall, well-made, and, on the whole, healthy-looking man, complaining of sickness, cough with slight expectoration, shortness of breath, palpitation, and swelling of the lower extremities. His face was somewhat pale, with perhaps just a trace of duskiness, and there was slight œdema about the ankles; the thyroid body was not enlarged, and the eyes were not prominent. At the time of admission it was noted that the heart was beating between 240 and 250 to the minute; that pulsation could be felt and seen over the whole præcordial region, and that the cardiac sounds, which were quite distinct, were free from murmur. The præcordial dulness was somewhat extensive, beginning in the third interspace above, and extending slightly to the right of the sternum. The apex impinged in the 5th interspace about half an inch outside the nipple. The lungs were resonant, and, excepting that on deep inspiration some finish crepitation was audible at the right base, the breath-sounds were healthy; expectoration slightly tinged with blood; respirations 32. There was no ascites or evidence of enlargement of abdominal organs. The urine had a sp. g. of 1025, and was free from albumen. Tongue clean, appetite good, bowels regular, temperature normal.

Dec. 5th, 2 P.M.—The patient has had several attacks of

fainting during the morning. They are said to have been attended with dyspnœa, and temporary stoppage of the heart, followed by over-violence of action. When I saw him the pulse at the wrist was irregular and impossible to count. On listening over the præcordial region the heart's action was found also to be irregular and very rapid. For periods of ten seconds or so at a time it was beating at the rate of 200 in the minute; then a strong beat would occur, and the pulse would drop for some seconds to a rate of 108; and again, after a short time, another throb would come, to be followed by renewed over-rapidity. This alternation of very rapid and comparatively slow pulsation continued during the whole time he was under observation. On one occasion the beats fell to 84. At 4 P.M. he was complaining of feeling faint, and his heart was beating at 250 in the minute. His breath was short.

Dec. 6th.—He had several attacks of sense of suffocation and faintness during last evening and night. They came on for the most part as he was dozing off, and prevented him from sleeping. About one o'clock in the morning he was in great distress; very restless, and wanting to get out of bed. He had half a drachm of aromatic spirits of ammonia and ten minims of tincture of digitalis given him, and became easier in the course of half an hour. The heart, when it was examined this morning, was beating at the rate of 164, and regularly. He has been complaining of nausea, and his feet are still œdematous.

Dec. 7th.—Passed a better night; had no return of faintness. When examined this morning the heart's beats were 182 in the minute; at half-past three in the afternoon they were 208. Cough better, expectoration less; urine sp. g. 1025; no albumen.

Dec. 9th.—Improving; but heart beating 224 in the minute.

Dec. 11th.—Seemed better all yesterday; but became poorly during the night; was restless, and suffered from nausea, sickness and diarrhœa. In the early morning the cardiac pulsations were 256; at 11 A.M. the cardiac pulse was declared by himself, and said by the sister of the ward, to be very slow. At noon, however, it had risen to 264. The respirations were then 28, and sighing; and the pulse could not be felt at the wrist; skin dry. *Midnight.*—Was sick at 4 P.M. During the evening has suffered from dyspnœa and palpitation; face now congested, lips and eyelids bluish; skin cool, perspiring; very restless and thirsty; heart's pulsations 256; respirations 28, and sighing; has spat a little bright blood. Ordered fifteen minims of tincture of digitalis.

Dec. 12th.—An hour and a half after taking the digitalis

he lost his lividity; felt easier, and inclined to sleep; but the heart's action remained rapid. Afterwards passed a fair night, and was tolerably comfortable in the morning. At 10 A.M. the cardiac pulse was 200; at 2 P.M. 220; the urine for the first time presented a trace of albumen. Ordered ten minims of tincture of digitalis, five grains of tartrate of iron, with infusion of calumba every four hours.

Dec. 14*th*.—The patient is much better; no recurrence of dyspnœa or faintness, and no return of hæmoptysis; no albumen in urine. He says that he feels "like his old self." But the pulse is still rapid. In the morning the heart's beats were counted at 168. At 3 P.M. the heart was again examined; it was then acting irregularly; at one time at the rate of 168, at another at that of 192, with short intervals of uncertainty.

Dec. 17*th*.—The patient has been going on well. The heart, however, has been acting variably, and, on the whole, rapidly. The cardiac beats have ranged from 120 to 132. At the present time the action is generally regular; but occasionally there are series of alternate strong and weak beats, the former only causing perceptible pulsation at the wrist.

Dec. 19*th*.—Doing well, and feels well. Pulse 60, regular and soft; no anasarca.

From this date until he left the hospital on the 5th of January he continued to feel in his ordinary good health; all faintness and dyspnœa, cough and anasarca, had disappeared; his appetite was good, his urine free from albumen, and he slept well. The heart's pulse was usually regular and about 60 in the minute; occasionally, however, it went down to 52, or rose to 78. On one occasion (December 21st) when I came into the ward the cardiac beats were only 34 in the minute: and the patient, though otherwise feeling well, complained of his heart thumping; consequently, the mixture containing digitalis, which hitherto he had been taking every four hours, was directed to be given thenceforth three times a day. On another occasion, while examining his heart (which was then acting regularly 60 times in the minute), I made him hold his breath for a few seconds, when immediately its action became irregular, presenting alternate strong and weak beats, at the rate (in combination) of 80 in the minute. The irregularity suddenly ceased after some seconds, and the rhythm and rate of movement returned to their previous condition.

When the frequency of the heart's action became permanently reduced, a soft systolic murmur was recognised at the apex. This had never been detected while the heart was acting rapidly, and was never observed to be absent when the organ was beating

slowly. It was perfectly distinct, but was inaudible in the back. All the other cardiac sounds were normal; and the second sound at the left base was not accentuated.

During the times at which the heart was acting rapidly, there was diffused pulsation over the præcordial region. This I examined particularly on two or three occasions, and came to the conclusion that the impulse corresponding to the surface of the right ventricle was synchronous with the apex beat. When the patient left the hospital, the heart's apex was situated just internal to the nipple. The radial pulse, excepting when the heart was beating slowly, was always very small and very feeble, and seemed to be irregular. It did not admit of being counted.

During the patient's stay in the hospital, his temperature rose on one or two occasions to 99, or a little more, and on one or two fell to close upon 95. It usually ranged between 97 and 98·6.

When he left he had gained flesh, and he looked, and (save for the presence of the mitral systolic murmur) might have passed for a healthy man.

After his discharge on the 5th, the patient remained quietly at home for a month or so, and then resumed work as a linen-draper's assistant, travelling every day three or four miles backwards and forwards between his home and place of business; and he came to my ward as an out-patient on several occasions. The following are the subsequent notes that were taken.

Jan. 7th.—Woke up at 6 A.M. yesterday morning with pain in the chest, and palpitation lasting about 10 minutes, and ceasing abruptly with a few forcible beats. Cardiac pulse counted immediately after he had walked up-stairs to the ward, and found to be 264, and regular. A little later it had risen to 288. Shortly afterwards, having rested for a while, the pulse had dropped to 84. Half a minute later it was 116, but irregular. After lying down for a short time it was 72, and regular. No murmur heard while the heart was beating rapidly. Pulsation diffused over whole cardiac area. Ferri Tart. gr. viij; Tinct. Digitalis ♏x; Inf. Calumbæ ℥j; t. d. s.

Jan. 18th.—Has had many attacks of palpitation both night and day, and there is a little return of œdema. At the present moment he feels well, but the heart is beating irregularly. Tinct. Digitalis ♏xv; ex haust.

Jan. 28th.—On the 26th, when getting into an omnibus, had an attack of faintness, lasting a second or two. Thinks he lost consciousness. But he has, on the whole, been much better than he

was previous to the last visit. Indeed, although his heart is beating at the rate of 220 in the minute, he is not conscious of palpitation, and feels and looks well.

Feb. 11th.—The patient has continued much better. But while under examination he complained of palpitation, and of a slight sense of faintness. During his visit the heart was acting regularly on the whole, but with extreme rapidity; on several occasions I counted the beats at 256 in the minute, and on several they varied distinctly between 300 and 308. During the examination he was standing up, and made no complaint. But on looking at him, his face seemed rather dusky, and I made him lie down. After a short time I examined him while he was still recumbent, and found his pulse at the wrist 64; the heart's beats being double that number, and alternately strong and feeble.

Feb. 18th.—Has continued to feel well, palpitation came on as he stood up to be examined, and the heart's beats were then 256 in the minute.

Feb. 23rd.—On this evening I got the patient to attend a meeting of the Neurological Society. He felt and looked perfectly well. His heart was beating regularly, and not rapidly, when he first entered the room. But soon the beats rose temporarily to about 240, and many of the members had the opportunity of verifying the fact. Several also of the members verified the presence of an apex systolic murmur when the heart was acting slowly.

After this he continued apparently very well. On the 26th (Saturday) he was engaged in business till midnight, and went home afterwards. The next morning (Sunday) he got up feeling quite well. He ate a good breakfast, and at one a good dinner; after which he went upstairs and played upon the piano. Suddenly the music stopped, and a fall was heard. On entering the room immediately afterwards, his friends found him lying on the floor dead.

Autopsy on the fourth day after death. Body much decomposed; some old pleuritic adhesions; lungs healthy; pericardium healthy; the heart generally was somewhat enlarged and dilated, and weighed $17\frac{3}{4}$ oz. Its muscular tissue was soft and flabby (doubtless from decomposition). The valves were perfectly healthy; all the cavities were nearly empty. Examined microscopically the muscular tissue showed no sign of degeneration.

There was no evidence of disease in any of the abdominal viscera.

The nervous centres were healthy, as were also the vagi and sympathetic cords in the neck and thorax.

Besides the above cases I have, since I have been on the look out for them, seen several which are obviously of the same class, but whose significance I should probably have formerly overlooked. The following is one of the most interesting and typical of them:

CASE VIII.—*Paroxysmal Palpitation apparently due to Strain.*

I was asked last February, by a medical friend of mine, to see with him the wife of another medical man whom he was attending. She had had excellent health up to ten days before, and had certainly never suffered from undue shortness of breath, or shown any signs of cardiac disease. At the time referred to, she was lifting a heavy box, when suddenly and quite without warning she experienced a sense of distress in the region of the heart, accompanied with faintness and oppression or difficulty of breathing. Since that time the attacks, lasting individually from a few seconds to a few minutes, have been of frequent occurrence, especially in bed. In bed, indeed, their constant and rapid recurrence has kept her awake all night. It was observed by her husband that during them her pulse was extremely irregular, and it seemed to him that (judging by the pulse at the wrist) the cardiac intermissions were frequently of several seconds duration. I received this history (which was subsequently confirmed) as my friend and myself were driving to the patient's house, and suggested to him that most probably these intervals of apparent inaction of the heart were really periods during which the heart was beating with extreme rapidity, though also with extreme feebleness. When I saw her she struck me as a well-nourished, healthy-looking woman; she had no difficulty of breathing, and was not in any physical distress. Her lungs were healthy and resonant. The præcordial dulness was of normal dimensions, and the heart's apex in the usual situation. Its sounds were healthy, save for the presence of a slight and soft systolic murmur in the aortic area; there was no evidence whatever of dilatation or enlargement of the heart; or (excepting the murmur) of any disease either in the valves or in the large vessels. There was no anasarca, and the urine was free from albumen; the pulse was regular, and about 100 in the minute. But during my interview with her, periods of regularity and comparative slowness of cardiac action alternated frequently with attacks (varying in length between 5 and 20 seconds) of marked irregularity. These generally began with one or two powerful beats, which were followed by beats at the rate of

between 200 and 250 in the minute. No pulse was perceptible at the wrist while the heart was acting thus rapidly. She had no pain at these times, but complained of a feeling of much distress. I saw the lady again at the end of April, and learnt that she had improved since I saw her, that for two or three weeks, while she was at the seaside, she had been almost entirely free from palpitation, and in her ordinary good health, but that during the last week or two (since her return to town) the attacks of palpitation had recurred. She had been taking liquor arsenicalis, but not iron or digitalis. She was at her best when I visited her, felt and looked well, was free from anasarca, had no dyspnœa, and the cardiac murmur, which I had recognised on the previous occasion, had disappeared. She had noticed latterly (as she had done before) that the palpitation came on chiefly at night time, and when lying down; and she lay on the bed while I was with her, in order to bring it on. But only two or three slight attacks of irregularity, lasting for a second or two at a time, occurred. She stated that when lying down she could arrest or prevent the palpitation by placing a cushion or pad under the loins, so arranged as to cause the back to be strongly arched backwards.

A case also, which I evidently formerly misinterpreted, has recently come under my observation in consultation with Dr. Clothier:

CASE IX.—*Paroxysmal Hurry of Heart of Fourteen Years' Duration—Death mainly from Bronchitis.*

This patient was an elderly lady living in Wales, whom I had known for 17 or 18 years. She had come up to London on a visit in the latter part of April of the present year. She was then in her usual state of health; but after a fortnight she seems to have caught cold, and to have contracted a sharp attack of bronchitis. I was asked to see her on the tenth day. At that time she was very ill, had much dyspnœa (her respirations were at the rate of 40 in the minute), had a frequent cough without much expectoration, and presented very slight anasarca. The urine was free from albumen. Her chest was resonant, but there was some crepitation at the bases. The cardiac pulsations were irregular, and 224 in the minute, the sounds being free from murmur, and the organ (so far as I could make out) of normal size. The pulse at the wrist was imperceptible. Her temperature was normal, and she was quite sensible. The case went on badly, and she died three days later of a combination of asphyxia and debility.

Between 14 and 15 years ago I was staying at her house in Wales, and was called up at night on one or two occasions, when she appeared to be suffering from what I then supposed to be angina-like attacks, and was, I thought, in great immediate danger. The heart was acting rapidly and irregularly, and I believed that she had serious organic cardiac disease. She recovered from these attacks; and I do not know (although I saw her on the average every year or two) that I ever examined her heart again. I heard, however, that she still suffered, from time to time, from similar attacks to those in which I attended her. But nevertheless she maintained fairly good health, and I was surprised to observe, year after year, that there was no aggravation of her cardiac symptoms. I saw her a little more than three years ago, and she then seemed well and cheerful.

I have no doubt, guided by my last examination of her, that her case from the beginning was a typical one of functional hurry of the heart, and that there never was any organic disease of that organ. Whether she would have died of her bronchitic attack, if her heart had been functionally healthy, may be questioned.

1. The cases just narrated possibly admit of arrangement in several categories. Thus, in some, the action of the heart was only extremely rapid; in some, it was rapid and also irregular; in most, the attacks of palpitation were paroxysmal; and in some the palpitation appeared to be persistent. Undoubtedly the most striking cases (and these first attracted my attention) were those in which phenomenal hurry of the heart, without obvious irregularity, occurred habitually in paroxysms of varying duration, in persons who were apparently in other respects healthy. A wider experience, however, has satisfied me that there is no essential distinction between the cases characterised by simple rapid cardiac action, and those in which rapidity and irregularity are combined, and that, although the tendency to intermission or remission of attacks is a common feature of the disease, the palpitation is always liable, sooner or later, to become indefinitely prolonged.

In those cases in which the palpitation occurs in paroxysms, these come on for the most part without warning, and often without obvious cause, last individually from a few minutes to several hours, or in some instances for several weeks or months, and usually end as suddenly as they began. The

intermissions are equally variable in duration, and, especially early in the disease, may be continued for many months.

In speaking of the rapid cardiac action as being in some cases regular, in others irregular, I have to confess that I use these terms relatively only. In the former class the cardiac pulse during the paroxysms may vary, roughly speaking, from 180 to 300 in the minute, even in the same case; but the changes from one rate to another are sudden, and the persistence of any one rate of beat is relatively considerable. In the other class, the cardiac pulsation is characteristically irregular; that is, small groups of slow and strong beats alternate with small groups of rapid and feeble beats, each group lasting between, say, one and ten seconds. The difference between the two cases is very obvious as a clinical fact, but the irregular pulse is apt at times to become regular, and by simple shortening of the duration of the successive rates of beat, it is clear that what seems a regular pulse would become irregular.

In all cases, as might be supposed, the pulse at the wrist is extremely feeble; and even in cases where the action of the heart seems regular, the regularity usually does not extend to the radial pulse. Indeed, when the heart is acting irregularly, the pulse at the wrist is often imperceptible for many seconds together, so that without examination of the heart it might be assumed that this was motionless during the whole of the time. In fact this disappearance of the radial pulse in the intervals between groups of stronger beats not unfrequently misleads the practitioner in this sense.

2. On what the undue rapidity of the action of the heart in such cases as I have narrated is primarily dependent, is very obscure. In most of my cases no definite cause for the commencement of the disorder could be discovered; and in those cases in which a cause was assigned, it was really doubtful if the apparent cause had anything to do with the matter. In Case VIII. the palpitation seems certainly to have been induced suddenly by over-exertion, and in Case VII. there is good reason to believe that the malady dated from an attack of cardiac distress brought on by running. In my 4th case there is no history of violent muscular exertion, but the palpitation first showed itself at a time when the patient had

much continuous work and responsibility. In another instance, Case V., the symptoms appear to have dated from what was said to be a sunstroke—an attack, the real nature of which is doubtful. In Case VI. there was a definite history of syphilis. On the whole, I am inclined to think that the evidence (such as it is) points to the origin of the malady in either mental or bodily over-exertion.

3. The question naturally suggests itself, as to whether these cases have any relation to hysteria on the one hand, or to exophthalmos on the other. In answer to the first question I may point out that most of my quoted cases, and, I may add, most of the other cases I have met with but have not thought it worth while to record, were in men; and that few, if any, even of the women presented any hysterical history, or manifested characteristic hysterical symptoms. As to the second question, I may observe that although extremely rapid action of the heart is one of the usual phenomena of exophthalmos, I have rarely or never observed such rapid action in that disease as has characterised the cases brought together in this paper, nor have I noticed in it the alternations between excited and normal pulsation which was the striking feature of so many of them. At the same time it is noteworthy that in Case III., in which the cardiac pulsations rose to 240 in the minute, there was actually a goitre. There was no additional evidence, however, of exophthalmos in her case; and I have reason to suspect that the association of the goitre with the palpitation was accidental.

4. One of the most remarkable facts in the history of cases of inordinately rapid action of the heart is the capability which many of the patients manifest of taking active exercise, and of doing their ordinary business even when the palpitation is upon them, and the little distress which the palpitation causes. This was noticeable even in the case of my first patient, who was kept in bed, and at rest, not because she felt ill or incapable of exertion, but because the medical man ordered it. Mr. S. (Case V.) was going about apparently well, and assured me that shortly before I saw him he had (although 65 years of age) run three miles in twenty minutes without suffering. Mr. C. also (Case VI.) not only went about his daily business,

but enjoyed a trip in Switzerland. The most remarkable example, however, of this phenomenon was Miss J., who up to the time at which her cardiac affection induced dropsy and other symptons of cardiac incompetence, lived during her attacks as she was accustomed to live when she was free from them, walked and did important official work daily, and even travelled to various parts of the country in pursuance of her duties as a Government inspector.

No doubt in all cases my patients suffered more or less from symptoms referable to the heart, and in some the symptoms were distressing and serious. They were mainly an uneasy feeling with a sense of fluttering in the præcordial region, shortness of breath, especially on exertion, and faintness; and associated therewith, more or less duskiness or lividity. Angina-like attacks occurred in one instance. One or two patients complained chiefly of a feeling of restlessness. And one (Case VI.), who always gave a very clear and graphic account of her condition, remarked over and over again that when the palpitation was on her she always felt irritable and in a hurry, and as though she must do two or three times as much work in a given time as usual. She, moreover, stated that she always experienced much distress at the onset of her attacks; but that this wore off in great measure after a short time, and that the sense of hurry and of fluttering in the region of the heart alone survived.

5. On the whole the results of my cases have not been satisfactory, and judging from them alone the prognosis of such cases is certainly not hopeful. In one or two the disease is still in an early stage, and what the event will be remains to learn. In two or three the disease has lasted for some years; and the attacks have shown a tendency to increase in frequency and duration. But I am sorry to say that, in large proportion, my cases have already proved fatal.

So far as I know, Cases I. and V. are still in progress; and the 8th case is up to the present time doing well, though not cured. Case III. was fatal, but it is possible that this was not a true example of the condition under consideration; and the 2nd case was also fatal, but the patient was suffering from advanced organic heart disease, with probably an aortic

aneurism, and his death may be fairly attributed to one or other of these lesions. All my other quoted cases have died. Miss J. (Case IV.) died of her disease after it had continued for fifteen or sixteen years. Mr. C. (Case VI.) died quite recently, having been liable to palpitation for about eight years. Mrs. J. (Case IX.) also died very lately, after suffering for fourteen or fifteen years. And A. W. (Case VII.) also died, probably at the end of about eleven years after the first symptoms of disease showed themselves.

Miss J. died with the ordinary symptoms of obstructive heart disease (namely, general anasarca, pulmonary apoplexy, and congestion of the liver and kidneys), which showed themselves for the first time about five weeks before her death, and were apparently induced by overwork at a time when her heart was acting rapidly. Mr. C.'s death was brought on in much the same way. His fatal symptoms, which included general anasarca, hydro-thorax (for which he was tapped twice), congestion of liver and kidneys, lividity and orthopnœa, appeared only about three weeks before he died. Williams died suddenly at a time when, so far as his feelings and appearance were concerned, he seemed to be perfectly healthy. Mrs. J. succumbed to what appeared to be a not very severe attack of bronchitis, the effects of which were obviously aggravated by the weakness of her heart.

Speaking generally of these cases of recurrent palpitation, I should be inclined to say, that the prognosis is fairly hopeful for those persons who are able to lead quiet lives, who avoid mental or bodily excitement and overwork, who protect themselves from catarrhal and other disorders likely to interfere directly or indirectly with the equilibrium of their circulatory organs, and who nurse themselves with care during their attacks of palpitation. And I am disposed to think that, in many such cases, the progress of the affection may be arrested. I base this opinion partly on the apparently complete restoration of such patients to health in the intervals between their attacks, partly on the long duration of some of their cases without the development of any serious cardiac and other complication, and partly on the circumstances of their fatal attacks when these at length happen to supervene. But it is

action. But that this is not so is obvious, both from the extraordinary feebleness of the pulse at the wrist, and from the very small amount of blood which is pumped at each ventricular systole into the arteries. The explanation is probably that the cavities of the heart are full of blood, and that the contractions of the ventricles are imperfect, so that only a small proportion of their actual contents is expelled at each systole. For if partial contraction occurs in a full but flabby and elastic-walled cavity, as the heart under such circumstances may be assumed to be, it is clear that the contraction in one part or zone must be attended with a general rise of pressure in, and consequent dilatation of, every other part, in which also there would be general synchronous pulsation. I believe, from having carefully examined several of these cases, that the apex beat and its impulse over the cardiac area are actually synchronous; whereas if, as one sometimes witnesses, and especially in cases of aneurism of the ascending arch, the pulsations of the surface of the ventricles and of the apex in a healthily acting heart are visible, they are distinctly alternate in rhythm.

7. As to the real nature of the disease which my paper is intended to illustrate, I have little to say. My belief is, as will doubtless have been gathered from all that precedes, that so far as the heart is concerned it is a purely functional disorder, that any actual cardiac disease which may be present in any case must be regarded as accidental, and that the slight hypertrophy and dilatation of the heart which may be found in patients who have suffered from the malady for years are (as I have already remarked) the consequence, and not the cause, of the palpitation.

8. I have also little to say under the head of treatment. In one or two cases during the attacks of palpitation, I have found digitalis, or digitalis combined with iron, of exceeding value; but in others these drugs have had no beneficial effect whatever. Digitalis and strophanthus, however, are remedies which would naturally suggest themselves, and are always worth a trial. If symptoms of obstructive cardiac disease become developed, as I have shown they are likely to do sooner or later, of course the ordinary treatment for such symptoms should be carried out. The little apparent suffering which

obvious that patients thus affected run many and continuous risks, and that death, either from a sudden faint or from the coming on of symptoms of cardiac incompetence or obstruction, is always imminent.

6. What the condition of the heart is in these cases is a matter of considerable interest. That such attacks of palpitation as I have described may be associated with organic heart disease is obvious, from the history of my second case, in which the patient was suffering from advanced aortic regurgitation combined with hypertrophy and dilatation of the left ventricle; and from a case now under my care in which the palpitation, coming on for the first time three or four months ago, is associated with the presence of a præsystolic apex murmur, referable apparently to the effects of an attack of acute rheumatism which occurred more than fifteen years ago. But in most of the cases I have seen there has been nothing to show that the heart was structurally affected; there has been no murmur, there has been no accentuation of the second sound at the left base, and the apex has impinged at the usual spot, or so near as it shows that there can have been little if any real change in bulk. In the only fatal case in which I have obtained a post-mortem examination, the heart was somewhat hypertrophied and dilated, but the valves were absolutely healthy, and so also was the muscular tissue. In this case a systolic apex murmur was recognised during life, when the heart was beating slowly, and appears to have been due (as was suspected during life) to the mitral valve allowing of regurgitation in consequence of the ventricular dilatation.

My belief is that the affection has no special connection with cardiac disease, and that dilatation and hypertrophy (when they occur in it independently of valvular mischief) are the slowly developed consequence, and not the cause, of the functional disturbance.

In connection with this subject of the condition of the heart, it is noticeable that during the attacks of palpitation the cardiac pulsations are visible over the whole præcordial region, and that there is visible throbbing in the large arteries of the neck. It might be hastily assumed that this extensive and apparently violent pulsation implied undue force of cardiac

ON RETROBULBAR INCISION OF THE OPTIC NERVE IN CASES OF SWOLLEN DISC.

BY ROBERT BRUDENELL CARTER.

I AM desirous to bring before the profession an account of an operation I have recently performed, by opening the sheath of the optic nerve behind the eyeball, in order to permit the escape of fluid in a case of great swelling of the termination of the nerve within the eye.

An operation for this purpose was first suggested by Dr. de Wecker, who, at the London meeting of the Quadrennial Ophthalmological Congress, in 1872, read a paper on the subject. He pointed out that the investigations of Schwalbe, continued by H. Schmidt, Manz, and others, had shown that the liquid contained in the arachnoidal space may in cases of exaggeration of intracranial pressure be forced through the optic foramen between the two coats of the optic nerve up to its insertion into the eye. Here the liquid, meeting an obstacle in the sclerotic ring, produces distension of the external coat on the one hand, and on the other, strangulation of the contents of the nervous sheath (fibres and vessels), which would explain the disturbance of vision, and the secondary atrophy of the nerve itself. Dr. de Wecker did not enter into any discussion as to the applicability of Schwalbe's theory in all cases, or as to whether the dropsy of the optic nerve is constant in every case of confirmed neuro-retinitis. He maintained it to be indisputable that in the great majority of necropsies the distension of the external sheath near the eye has been met with, and that Schwalbe's theory affords the most satisfactory explanation of the production and symptoms of neuro-retinitis. According to this theory, he considered that there were two indications to be fulfilled: in the first place, to give issue to the accumulation of the cerebral liquid by making an incision into the external coat of the optic nerve; and, secondly, to relieve the strangulation of the nerve by incising the sclerotic ring where it forms the junction of the sheath with the external enveloping membrane of the eye. By proceeding thus, he hoped to relieve the symptoms of compression, not only of the nerve itself, but those of the

many patients experience during their attacks permits and encourages them at such times to go about their ordinary work and even to undergo considerable mental and bodily exertion. It is clear, however, that all such labours are detrimental, and attended with danger; and that the patients should be kept at rest. When one considers the tendency there is in these cases for their malady to become more and more grave and intractable as time goes on, the importance of treating them in the intervals between the attacks, and especially in the earlier stages, becomes obvious. I am not prepared to recommend at such times the employment of any particular drug, but it seems to me that our aim should be to maintain the patient's general health, to prevent as far as possible undue mental or emotional exertions, or severe muscular strain, or continuous mental or bodily hard work, and to treat with special care every attack of palpitation.

cerebral centres—in other words, to remove the pain and inconvenience arising from the excess of intracranial pressure.

Dr. de Wecker went on to say, that he had performed the operation in two cases of cerebral affection (probably tumours of the cerebrum). In one of the cases, a man of 40 (whose right eye was operated on), neuro-retinitis was completely regressive in the right eye, and in the other regressive also. There was only a slight degree of vision, scarcely allowing the patient to do any work, even if the weakness of the legs, with which he was at the same time affected, had permitted it. The other patient, a woman of 30 (whose left eye was operated on), was completely blind, and was at the same time the subject of paraplegia, partial paralysis of the left facial nerve, and great difficulty of moving the tongue. She also suffered from incessant headache. The neuro-retinitis was in this case evidently regressive in both eyes, and especially in the left.

Dr. de Wecker described his operation by saying that he made an incision between the rectus externus and the rectus inferior muscles, at a distance of one centimetre from the margin of the cornea. Then, cutting the conjunctival and sub-conjunctival tissues, he penetrated between the eye and Tenon's capsule with a pair of probe-pointed scissors until he reached the optic nerve. He then introduced a spatula, and luxated the eye upwards and inwards. It was easy after the luxation of the eye to feel with the spatula the distended nerve, and to introduce a sheathed neurotome, an instrument made by Mr. Mathieu for the purpose. It consists of a stem curved to fit around the eyeball, terminating in a notched extremity intended to feel for and to embrace the nerve, and concealing a sheathed blade. Dr. de Wecker believed the mechanism of this instrument to be such, that by slight pressure on the spring only that part of the nerve would be cut with which the notch was placed in contact, at a distance of one centimetre behind the eye. The operator was to incise the sheath of the optic nerve and the sclerotic ring, moving and pressing the instrument from behind forwards. He would then remove the instrument, after having pushed the spring back, so that the blade could not cut more than was desired.

Dr. de Wecker stated the results of his two operations as follows:

1. There was no pain after the operation, which was performed without the aid of anæsthetics. 2. Very great relief of the headache, especially on the side operated upon. 3. On removing the bandage, twenty-four hours after the operation, there was a very slight congestion of the eyeball, and the pupil deviated to the opposite side of the penetrating wound, probably in consequence of rupture of the ciliary nerves.

This deviation of the pupil disappeared in a few days. 4. The operation having been performed in complete regressive forms of neuro-retinitis, no amelioration of sight was to be expected; nevertheless the male patient seemed to have improved in the operated eye, for in a week he was able to perceive the light. The beneficial result in this case seems to have been indisputable, and the medical men who assisted at the operation were of the same opinion, as there was a marked influence on the headache, and on the general state of the health. The male patient was able to stand more firmly on his legs, and to answer questions promptly. He seemed delighted with the operation, on account of the general relief he had experienced, although he had gained nothing as far as sight was concerned. The result of the operation was less manifest in the female patient, who was only relieved of her headache.

The ophthalmoscope did not discover the incision, which was perhaps not made deeply enough; but there was a slight increase in the volume of the vessels, especially of the veins.

In September 1872, Dr. de Wecker's experiment, if I may so describe it, was repeated by Mr. Power on the right eye of a girl of 13, this eye having been blind for two years, and having still some swelling of the optic disc. Mr. Power has favoured me with the notes of the case; but it is sufficient to say that, beyond a doubtful increase in the power of perceiving light, no beneficial effect was obtained.

Dr. de Wecker's paper greatly impressed me when it was read; but I could not escape from the conclusion, that a case of advanced optic-nerve atrophy, although possibly suitable as a *corpus vile* on which to demonstrate the practicability and harmlessness of the operation, was not one in which any good result could reasonably be expected. I felt, moreover, that the mode of procedure was extremely faulty, and that the structures concerned were far too important to be incised without the aid of sight. The least obliquity of direction of the unsheathed blade might cause it to divide one or more of the ciliary nerves; and, even if the blade were placed with diagrammatic correctness, the smallest excess of penetration might wound the central retinal vessels in the nerve trunk. The attempt to extend the incision through the sclerotic ring seemed to be especially perilous, and likely to lead to puncture of the choroid and to extravasation of blood within the eye in the neighbourhood of the macula lutea. In short, the operation, as described by Dr. de Wecker, appeared to me to be too uncertain and too dangerous to be justifiable in any circumstances, although I had no doubt of the soundness of the principle on which it was based.

During the last few years it has been my lot to see a large

number of cases in which, in connection with the growth of intra-cranial tumours, or with other morbid processes, there has been great swelling of the intra-ocular extremities of the optic nerves. In many cases of this kind sight is at first absolutely unaffected, the swelling being limited to the connective tissue of the disc and of the fibre layer of the retina, and being apparently produced by a moderate amount of mechanical impediment to the circulation, not enough either to arrest conduction through the nerve fibres, or to close the channels of the vessels. In some instances the swelling of the optic discs has disappeared under treatment, and complete recovery has ensued. In others, nerve atrophy and blindness have been produced; and these consequences may, I think, be explained in two ways. If I may dwell for a moment upon the anatomy of the parts concerned, it will be to remind the reader that the optic nerve is closely invested by a sheath derived from the pia mater, and this again, but loosely, by a sheath derived from the dura mater. Between these two sheaths there is an interval, or inter-vaginal space, which terminates in a cul-de-sac at the level of the back of the eyeball, where the dural and pial sheaths unite to blend with the sclerotic. This inter-vaginal space contains membranous processes derived from the arachnoid, and communicates with the sub-arachnoid space; so that, as was shown by the authorities cited by Dr. de Wecker, an excess of sub-arachnoid fluid will make its way into the intervaginal space, compressing the nerve trunk and distending the dural sheath, which, especially in the immediate neighbourhood of the eye, has often been found to be much dilated. The central artery and vein of the retina pierce the sheaths at a point which is from fifteen to twenty millimetres behind the eye: and it is manifest that compression of the lower part of the nerve trunk, in which the vein is still contained, must impede the return of blood, and must occasion dropsical swelling of the nerve termination within the eye, and of the adjacent retina, with turgescence of the retinal branches of the veins. The effect of such compression must depend upon its degree, and experience shows that it may be sufficient to produce distinct dropsical swelling, and that this may continue (within my own knowledge for more than three years) without any affection of the sight. On the other hand, the compression of the nerve trunk as a whole may increase so rapidly and to such a degree as quickly to destroy vision by arresting conduction through the fibres; in which case it must also arrest the flow of blood through the vein, an occurrence usually attended by hæmorrhages into the retina. When the compression increases more slowly, the consequent dropsical effusion may excite inflammation of the connective tissue of

the optic disc by mechanical disturbance of its structure; and this inflammation may in its turn give rise to an interstitial plastic exudation, which will gradually contract, and will produce atrophy of the disc by strangulation of its capillary vessels and of its nerve fibres. With the commencement of contraction vision begins to suffer, and is ultimately completely extinguished.

It is very common to hear all cases of swelling of the intra-ocular termination of the optic nerve described as "optic neuritis," but I am strongly of opinion that this description is erroneous. I am only too familiar with optic neuritis; and the first symptom of its existence is great impairment of sight, which, if the inflammation be not relieved, speedily passes into blindness. The neuritis is usually retro-bulbar in its origin; and some days may elapse, and central vision may be almost lost, before any evidences of exudation can be discovered in the disc. Even then, such evidences are often so slight that only a practised eye would detect them; and I presume that the part of the nerve primarily affected is in such cases posterior to the point at which the retinal vein emerges from the sheath, and that hence no mechanical or dropsical swelling of the disc is produced. I do not believe in the possibility of any true neuritis without marked derangement of the function of the affected nerve; and when I see, as I have often seen, considerable swelling of the optic nerve within the eye, continuing for weeks, months, or even years, without any injury to the sight, I feel convinced that such swelling is not neuritic, but only passive or dropsical, a result of impeded circulation. I have no doubt that dropsical swelling may excite a sort of spurious neuritis, in the way already mentioned, by the disturbance which the swelling occasions, but I regard this effect as being confined to the connective tissue, and not as constituting neuritis in any true sense; nor do I think that the mere presence of leucocytes in the optic disc affords evidence of any action to which the word "neuritis" can properly be applied. We have all seen cellulitis of the leg; and we have all seen a certain amount of inflammation, attended by erythema, as a consequence of dropsical swelling; but we should not describe a limb which presented the latter conditions as an "inflamed leg," even though it would no doubt contain leucocytes in abundance, and though the fluid in the meshes of the connective tissue were coagulable and plastic in its character. Notwithstanding all this, we should know that a few judiciously placed punctures, by allowing the fluid to escape, would speedily produce a change for the better; while, in actual cellulitis, not even free incisions would generally prevent destruction of tissue. To confound the two

conditions, on account of the presence of microscopic products common to both, would be to permit histological refinements to outweigh the evidence of broad clinical facts.

The cases to which I am now chiefly desirous to attract attention are those in which, after the persistence of optic nerve swelling for a certain time, without impairment of vision, such impairment at last commences, and proceeds more or less quickly to complete blindness. In some of these cases the loss of sight may be due to the development of a tumour or other intercepting lesion between the optic nerves and the cerebral centres of vision; but more frequently, in my judgment, it is due to one of the two causes I have mentioned, that is, either to rapid compression of the nerve trunk as a whole by increase of fluid within the dural sheath, or to slow strangulation of the capillaries and fibres by the contraction of an interstitial effusion of a plastic character. In some of these cases the primary lesion may be recovered from, in others it may not destroy life for a long period; but in both the patient is left blind.

I may mention, by way of illustration, the history of a gentleman who first consulted me on the 30th of September, 1885. He had then great swelling of both optic discs, but scarcely any affection of the sight. He continued much in the same state until the following April, when his sight began to fail, the previously existing swelling passed into atrophy, and in a few weeks he became totally blind. There is reason to believe that he has an intracranial tumour; but he is still in possession of his mental faculties, and although reduced in strength, has no paralysis. In thinking over his case, I determined that I would take the next opportunity of a similar kind to open the nerve sheath behind the eye, in the hope that the evacuation of the contained fluid would preserve the nerve from atrophy and the vision from destruction. Such an opportunity was afforded by the case which forms the subject of this paper, and which I will now proceed to relate.

E. O., a lady's-maid, 26 years of age, was brought to me by her mistress on the 18th of November, 1886. She had been somewhat out of health for a fortnight or so, had recently been exposed to cold when driving, and had received a blow, of no great severity, on the right parietal region of the head; but there was nothing very definite in either her history or symptoms. She had suffered for ten days from a good deal of aching pain across the forehead, and also in the occipital region, but her chief complaint was that, when rising on the morning of the 9th, she discovered that she saw imperfectly with the left eye. She could distinguish objects, but said that they looked dark and dim, and this state of the sight, although

varying a little from time to time, had remained substantially unaltered. Central vision of the left eye was found to be equal to about two-thirds of the normal standard, but the eye was blind as to the temporal half of its field of vision, and the field was much contracted in other directions also, as shown by the chart in Fig. 1, which was taken on the 26th of November. The left optic disc was swollen, and its margins were entirely concealed, the swelling encroaching upon the retina in an unusually broad zone of circumneural turbidity, through which not even the colour of the choroid could be seen. The central part of the swelling showed numerous small hæmorrhages, and its apex was best defined with a convex lens of four

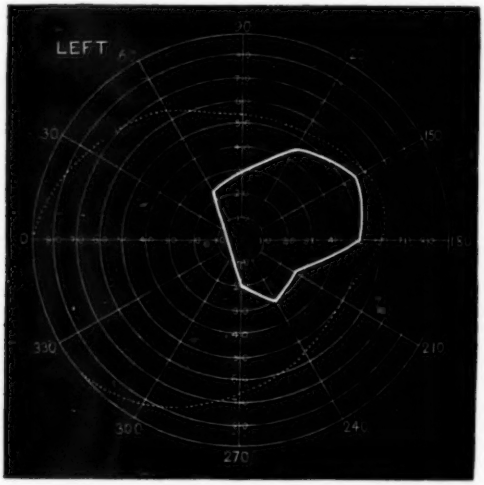


FIG. 1.

dioptres, which, allowing for one dioptre of hypermetropia, pointed to an actual elevation of one millimetre. Beyond the swelling, the retinal veins were distended and tortuous. The right eye was normal in aspect and vision, and had one dioptre of hypermetropia. There were no general symptoms of a kind to throw any light upon the essential nature of the lesion. The tendon reflexes were active, and there was a trace of ankle clonus.

The patient was admitted into the National Hospital in Queen Square, under the care of Dr. Hughlings-Jackson and myself, and she was placed upon iodide of sodium with mercurial inunction. She very soon became mercurialised;

but the only other obvious change in her condition was that the swelling of the optic disc, and the breadth of the turbid circumneural zone, continued steadily to increase. Some opaque white patches appeared among the effusion, a considerable hæmorrhage occurred over the position of the inner margin of the disc, and on the 17th of December, flocculent opacities in the vitreous were observed over the apex of the swelling.

The vision was at this time unchanged, but I felt that it was exposed to great risk from increasing pressure or from interstitial strangulation, altogether independently of any unknown lesion by which the origin of the local condition might be explained; and I determined, with the assent of Dr. Hughlings-Jackson, to repeat Dr. de Wecker's operation, if possible, in a more certain and satisfactory way. I convinced myself, by trial upon the dead subject, that the insertion of the optic nerve could only be exposed from the outer side of the orbit, and, after much consideration, I determined on the course about to be described.

On the 28th of December, the apex of the swelling being then best defined by a convex lens of nine dioptres, showing an actual elevation of close upon three millimetres, or twice the diameter of the disc itself, the patient was placed under chloroform, and the lids were widely separated by a rack speculum having its bar on the nasal side. I divided the conjunctiva and sub-conjunctival tissue in a vertical line, about a centimetre from the corneal margin, so as to expose the external rectus muscle a short distance from its insertion. I passed two strabismus-hooks under the muscle, and then, being provided with two threads of fine carbolised catgut, each carrying a needle at either end, I passed the two needles of one thread through the tendon, from within outwards, between its insertion into the sclerotic and one of the hooks, and the two needles of the other thread, also from within outwards, between the belly of the muscle and the second hook, and then divided the muscle between the hooks. The eyeball was next rotated inwards, while the orbital portion of the divided external rectus was lifted and turned outwards. By a succession of small scissor snips, I carefully divided the capsule of Tenon, and other resisting structures, to the necessary extent, and presently succeeded in bringing the insertion of the optic nerve into view. I had furnished myself with a fine sharp-pointed knife, and with a very delicate sharp hook, each mounted on a flexible platinum stem. I picked up and steadied the nerve sheath with the hook, and incised it with the knife, in the direction of the axis of the trunk, for perhaps a quarter of an inch, up to the insertion into the eye, but

without dividing the sclerotic ring. An escape of fluid showed the division of the sheath to be complete. As soon as this was seen, the eyeball was replaced, the parts of the divided external rectus were brought together by the sutures already in position, and the conjunctival wound was united by two others. Both eyes were closed and bandaged, and the patient was put to bed.

The operation was followed by no discomfort. There was no rise of temperature, the muscle united readily, and the external wound showed no irritation. On the next day the patient was somewhat nervous and excited, but on the third day she said that she felt quite well, and that she was completely relieved of the pain and "muzzy feelings" in the head by which she had for some weeks been troubled.

A mere glimpse with the ophthalmoscope, four days after the operation, showed that the swelling of the optic disc had diminished, but no complete examination was made until the 7th of January. By that time the broad zone of circumneural retinal opacity had disappeared, and the margin of the disc was visible around rather more than the outer half of its circumference. The best definition of the apex of the swelling was obtained with a convex lens of four dioptres, showing that the actual prominence had diminished by about one-half, or to what it was when first examined.

From this time forward the subsidence of the swelling, although not rapid, was steadily progressive. The remaining blurring of the inner margin of the disc has disappeared; and all the extravasated blood has been absorbed. The vessels of the disc are visible throughout the whole of their course, and there is no longer any impediment to the venous circulation. There is about one-third of a millimetre of remaining swelling, and the general aspect of the surface is still somewhat veiled and abnormal, but it seems to be in course of steady restoration to the natural condition. If I had divided the sclerotic ring, the recovery might possibly have been more rapid.

The acuteness of central vision has become normal, and early in March the field began to increase on the previously blind temporal side. The chart in Fig. 2 was taken on the 21st of March, in a very bad light, and the field has since undergone further extension.

The movements of the eyeball, and those of the pupil, are perfect, and there is nothing to show that any surgical interference has taken place. The general condition has lately undergone steady improvement, and there is, I think, reason to hope that complete recovery will occur.

The issue of this case seems to me to establish that the

operation devised by Dr. de Wecker, and which, as he endeavoured to perform it, I should not care to attempt, has been brought within the limits of safe and prudent surgery. The case shows that the sheath of the optic nerve can be exposed to view, that any fluid which it contains can be evacuated by incision, and that this can be accomplished without risk to the patient, or without the possibility, assuming the exercise of due care and skill, of any injury to the eye. If thus much be conceded, we have only to inquire in what circumstances, if in any, such a proceeding is likely to be advantageous to a patient.

For the purposes of such an inquiry, I should in the first

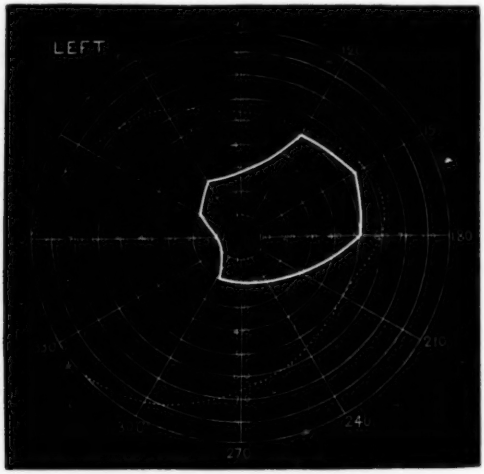


FIG. 2.

place exclude all cases in which swelling of one or both optic discs is apparently due to the presence of an intracranial tumour which can be localised and removed. Mr. Horsley's operations have shown that, in such circumstances, the removal of the tumour itself is followed by the subsidence of the disc swelling; and the results of the major operation would render the minor one unnecessary. But, apart from these cases, I think incision of the nerve sheath should be practised whenever swelling of one or both discs is attended by the commencement of impairment of vision. Such impairment might be caused, in some instances, by a lesion unconnected with the sheath, which its incision could not touch, but it would more

frequently be caused by external compression or internal strangulation of the nerve, and these conditions might certainly be relieved. It is well known that many cases of swollen disc go on to atrophy and blindness while the patient recovers, or at least does not die; and I feel strongly that, in most of these, sight might be preserved by the means to which it has been my object to direct attention.

We also see instances of loss or great impairment of sight, from nerve atrophy after various acute diseases, fevers, the exanthemata, and others, and in these we generally find ophthalmoscopic evidence that the atrophy has been preceded by swelling, probably as a consequence of some meningeal effusion. If it were the custom to watch the optic discs in all grave cases of this kind, especially on the occurrence of head symptoms, the commencement and the increase of swelling would be observed, and incision of the nerve sheath would not improbably preserve the sight in many instances.

I should by no means be without hope, moreover, of obtaining good results from the operation in a certain proportion of cases of real or primary optic neuritis, those of which I have already spoken as being speedily destructive to sight, and as being attended with a very small amount of disc swelling. In these, I apprehend, the neuritis is not only retro-bulbar, but also posterior to the emergence of the retinal vein; and it is highly probable that the inflamed and swollen nerve is compressed and injured by its unyielding sheath. Assuming genuine neuritis to exist, and the nerve to be suffering compression in the manner supposed, I cannot doubt that the relief of the compression might lead to the resolution of the neuritis; and I should be quite prepared, in any such case, to undertake the operation. I should introduce, through the first aperture in the sheath, a fine probe-pointed knife between the sheath and the nerve trunk, and should run this knife as far back as possible towards the apex of the orbit, in the hope of making an incision sufficiently long to relieve compression over the whole of the inflamed portion of the nerve. In the indicated conditions, and possibly also in others, I cannot but think that the operation I have described has a fair prospect of usefulness; and I beg leave to commend it to the attention of all those who are likely to have opportunities of testing it in practice.

Clinical Cases.

CASE OF MYXO-FIBROMA OF THE FIFTH DORSAL NERVE EXTENDING ON TO THE SPINAL CORD.

BY J. MITCHELL BRUCE, M.D., F.R.C.P.,

AND

FREDERICK W. MOTT, M.D., M.R.C.P.

GEORGE L., aged 37, by occupation a game-keeper, was first seen by Dr. Bruce on May 7th, 1886, for incomplete paraplegia with impairment of sensation as high as the epigastrium, some difficulty of urination, and constipation:

He complained of a boring pain in the mid-dorsal region, radiating into the left axilla, and of a feeling of numbness in the left lower limb, extending upwards. The pupils were equal, and reacted both to light and accommodation. The knee-jerk was well-marked on both sides. Physical examination of the chest revealed nothing abnormal in the condition of the thoracic viscera, nor was there any evidence of disease of the abdominal organs. The patient was recommended to go to Charing Cross Hospital, and on May 9th he was admitted under the care of Dr. Bruce. The following notes were taken:—

With the exception of a brother having died of phthisis, the *family history* was good. He was married when 27 years of age, and has had four children; one only is living. There was no history of syphilis. Until four years ago, patient had been in robust health. Many years ago, he had had a fall on his back; but he did not think that there was any permanent injury resulting from it, for many years elapsed between this and the period when symptoms of his complaint appeared.

It was about four years ago that he noticed a sharp pricking pain in the fifth intercostal space of the left side, just inside the nipple. This continued, and, after a month or so, became more severe and stabbing in character. For about three years this pain persisted; but he was able to continue his

work until Christmas 1885, when, on account of the great pain he experienced in walking, he was obliged to give up his occupation as a game-keeper. The pain was particularly severe if he bent down; for example, to tie his boot-laces.

Just after Christmas he noticed that his legs swelled, and the following March the left foot began to feel numb, the numbness gradually spreading up the left leg. He noticed this also, though to a much less extent, in the right foot. On account of this numbness in his feet, patient says he "blundered about." In less than a week after the numbness had set in, he noticed that his legs, which were usually extended during sleep, often became flexed. He said that he had felt weakness for some time in his left leg, and since Christmas he had been troubled with constipation and difficulty in micturition. He has had no priapism nor loss of sexual power. The reason he sought admission to the hospital was for loss of power and numbness in his legs. He believed that the pain he had suffered for four years had little to do with his present illness, having been told by the doctor it was rheumatism.

May 12th.—The patient now complains of numbness in the *right* lower limb, increasing in degree. Plantar reflex distinct; cremasteric and abdominal reflexes absent; knee-jerk increased, especially on the left side. Ankle-clonus on the left side.

May 15th.—Patient complains of pain in the mid-dorsal region which radiates round the thorax, especially on the left side. Occasionally the pains shoot down the left thigh; these pains are particularly severe at night. There is a girdle sensation at the level of the fifth intercostal space. Absolute anæsthesia and analgesia exist over the lower limbs, and on the trunk up to the level of the umbilicus. Power of movement of lower limbs is now completely lost on both sides. Occasionally there is spasmodic flexion.

Reflexes.—Plantar reflex still present; cremasteric and abdominal, lost; epigastric, present; knee-jerk, lost; ankle-clonus, now absent.

There is complete loss of control over the rectum, retention of urine, and unconsciousness of rectal and vesical acts. Œdema of the lower limbs, and bedsores on the hips.

May 28th.—Condition carefully investigated. Anæsthesia as high as the eighth rib; above this to the fifth intercostal space is a zone of hyperæsthesia. On the right side, there is anæsthesia as high as Poupart's ligament. Painful sensations are lost as high as the margin of the ribs on the left side, as high as the middle of the thigh on the right side. Sensations of heat and cold are not appreciated on either side

below the level of the umbilicus. Plantar reflexes, exaggerated; slight ankle clonus on left side; knee-jerks, absent; cremasteric, epigastric, and abdominal reflexes all absent.

Muscles.—Right limb: can flex and extend ankle, knee, and hip joints, but there is considerable weakness. Left leg completely paralysed at all the joints except the toes, where there are slight voluntary movements. Occasionally the legs extend themselves; and sometimes patient finds them flexed on waking. No rigidity exists at any of the joints. There is slight redness and swelling of the left knee-joint, with some effusion. The muscles of the limbs are not wasted; and although requiring a stronger current than normal to excite contraction, there are no qualitative changes.

On examination of the spine, no deformity could be ascertained; but there was tenderness over the mid-dorsal region. The temperature has been always normal, and physical examination revealed no organic disease of the heart, lungs or abdominal organs.

The patient still suffers from constipation, and difficulty in evacuating his bladder.

An attempt was made to relieve the pain by blisters and fomentations, and he was placed upon iodide of potassium, ten grains three times a day, on the possibility that he was suffering from compression of the cord by a gumma. He remained very much in this condition, the pain perhaps being a trifle better, till June 11th, when he suffered great pain in the hypogastrium. The water was drawn off, but after some days the urine became ammoniacal. From this time until his death, the bladder was washed out daily with one in twenty solution of boro-glyceride.

June 25th.—Morphia injections commenced on account of the pain. He is now quite unable to move the right leg, and feels tickling sensations in both limbs. It is now necessary to draw off all his water.

July 9th.—Ankle-clonus is now very marked on both sides. Plantar reflexes are exaggerated, but the knee-jerk on both sides is still absent; also the abdominal and cremasteric reflexes. Anæsthesia and analgesia now at the same level on both sides. The rectum is only evacuated by enemata. He complains of a constant sense of fulness of the belly; but there is no vomiting. Pupils are normal, and the upper extremities are unaffected.

August 18th.—Patient is lying in bed on his back, complaining of a great deal of pain on the right side, which he says is greatly aggravated by twitching in his legs. For this reason he lies with his legs semi-flexed, not that he has any

power over them, but he draws them up into this position with his arms. On attempting to straighten the knee-joints, great rigidity was observable.

Ankle-clonus extremely well marked. Both feet are swollen and œdematous. The legs do not appear wasted.

The patient remained in this condition several months, daily becoming more exhausted by the pain, which was only relieved by continual hypodermic injections of morphia.

October 3rd.—Began to pass fæces unconsciously. A few days later the bedsores spread rapidly in area; and he eventually died November 15th, 1886.

Necropsy, twelve hours after death. Body emaciated; bed-

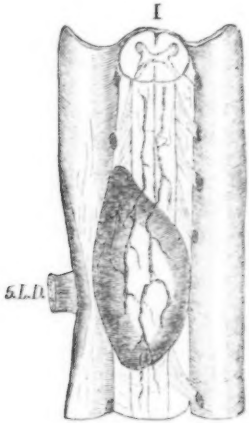


FIG. 1.—Tumour and infiltrated 5th left dorsal nerve, as seen on slitting up the dura mater. The part of the tumour in which there is no shading represents the thin part of the cyst-wall. The posterior spinal vessels run upon the surface. Natural size; somewhat diagrammatic.

sores at all the points where pressure occurred. A large sore, quite a foot across, exists over the buttocks, sacrum, and hamstrings, the muscles and bone being exposed. The skin had sloughed about the groin and the lower end of the left thigh, where pressed upon. There are also sores over the shins and heels.

Abdomen.—Superficial view normal. Liver much enlarged, weighing 6 lb. 6½ oz.; pale yellow, the edge rounded; no albuminoid change. Nothing else noteworthy in abdominal organs.

Thorax.—Pleuræ normal. R. and L. lungs healthy. Heart-substance pale; otherwise normal.

Brain—quite healthy.

Spinal Cord.—When the spinal canal was opened in the mid-dorsal region, the theca vertebralis was found swollen by a bluish mass within it. The dura mater having been slit up along the posterior surface, an ovai swelling (see Fig. 1), about 1 inch long by $\frac{1}{4}$ inch thick, was found lying upon the spinal cord and the left 5th dorsal, which could be seen entering it. The tumour was cystic, and on section was found to contain a blood-stained fluid. There were large vessels, continuous with the spinal vessels, running over its surface. The nerve was swollen to three times its natural size; and was soft, red, and gelatinous, being apparently infiltrated with some new growth. It may be mentioned that a careful search of the body was made for secondary deposits, but without positive results.

A portion of the infiltrated nerve and the spinal cord were

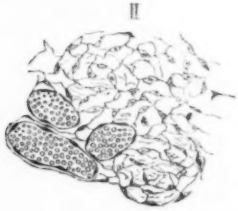


FIG. 2.—Section of a portion of the infiltrated nerve, showing branched, stellate, and spindle cells forming a reticulum with dilated vessels. Magnified, 200 diameters.

hardened in two per cent. solution of bichromate of ammonia. Sections of the former obtained by the paraffin method (Fig. 2) showed the growth to be a myxo-fibroma, which had apparently resulted from reversion of the connective tissue of the nerve to the embryonic type. It was very vascular, the vessels appearing to be dilated capillaries. The nerve was almost entirely destroyed; there were, however, a few small bundles of partially atrophied and degenerated nerve fibres left, but even here there could be seen a conversion of the connective tissue, forming the perineurion and endoneurion, into mucoid tissue. The nerve when traced up to the cord was seen to extend by the posterior root into a cyst.

Microscopical examination of the cyst—showed a fibrous capsule, embedded in which were numerous dilated vessels, suggesting the possibility that rupture of one of them had contributed to its formation. Fig. 3 shows the cord lying

beneath; and although this is completely disorganised by compression, the cyst is seen to be situated outside the pia mater, and the cord is in no way *directly* involved by the growth.

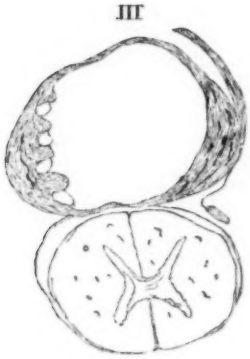


FIG. 3.—Transverse section of the cord and cyst, magnified 3 diameters. The vascular nature of the walls of the latter is obvious. The cord was disorganised in this situation, and the position of the grey matter is represented by a space corresponding to it in form. It will be observed that the tumour lies entirely outside the cord.

Examination of the cord, after staining by Weigert's method, exhibited the following degenerations:

1. In the *cervical* region: Posterior median, direct cerebellar, and ascending antero-lateral columns (Fig. 4, p. 216).

2. *Below* the *tumour:* crosses pyramidal tracts on both sides (Fig. 5, p. 216).

There were no changes in the multipolar cells of the anterior cornua; but apparently, from the congested state of the vessels in the posterior cornua, the grey matter in this situation might have been affected by inflammatory changes.

Comments on the Case.—There are many points of interest in this case.

1. The history points to the growth having commenced in the nerve, and having extended to the spinal canal after about four years, the patient all this time attributing his suffering to rheumatism.

2. The history and symptoms thereafter indicate a slow but increasing compression of the cord; and this led to the diagnosis of tumour. As the patient was a young man, cancer could be excluded. The diagnosis then lay between syphilitic growth, vertebral caries, tuberculous growth, some form of

sarcoma, or an uncommon non-malignant growth. *Syphilis* was improbable, because there was no history pointing in that direction, nor were the symptoms in any way relieved by anti-syphilitic treatment. *Vertebral caries.* There was no history of injury nor cause for caries, and for four years there had been little or no tenderness over the spine, everything pointing to an affection of the nerve. Moreover, very careful physical examination failed to discover any of the ordinary signs of disease of the spine.

Tuberculous tumour and sarcoma were improbable, because of the very slow course of the symptoms, and the complete

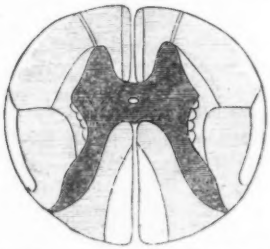


FIG. 4.—Diagram showing degenerated tracts (dotted regions) above the tumour.

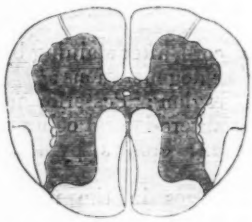


FIG. 5.—Diagram showing degeneration of crossed lateral tracts below the tumour.

absence of any evidence of disease in the internal organs. Thus by a process of exclusion we might conclude that the evidence was strongly in favour of a *non-malignant growth*, spreading from the nerve or its root on to the spinal cord.

3. Tumour of a non-malignant nature having been diagnosed, we now greatly regret that an attempt was not made to remove it. On one occasion the patient expressed his willingness to submit to an operation. Had we called in a surgeon, there is but little doubt that the tumour might have been removed successfully, for it proved in no way to have invaded the cord. Mr. Victor Horsley, who has had such success with

operations on the brain, has expressed the opinion to us in conversation, that this was in every respect a case that would have been favourable for operation.[1] Dr. Gowers, in his recent work on the spinal cord, says: "It is highly probable that surgery may be able to cope, in some degree, with meningeal tumours. Modern methods render the opening of the spinal canal far less formidable than it formerly was, and the removal of a tumour from the membranes of the cord would involve less immediate danger of serious consequences than the removal of a tumour from the brain." Although myxomata of nerves are not uncommon, and those commencing in the membranes of the spinal cord not very rare, yet such a one as this, commencing in the nerve or its root and spreading on to the spinal cord, is—as far as we have been able to ascertain—unique. Notwithstanding this, we have published an account of the present case, not only from its own inherent interest, but because it appears to us to justify the belief, that instances do occasionally occur in which tumours compressing the spinal cord may be treated surgically with complete success.

4. There are some other points of interest in the case in connection with the symptoms: (1) the absence of all the reflexes except the plantar; and (2) loss of movement in the left limb, and eventually the right limb, except slight movements in the toes. Dr. Bastian, in his article on diseases of the spinal cord in Quain's "Dictionary," has noted a similar fact in a case that occurred in his practice, and he has attributed it possibly to vaso-motor spasm of the vessels in the grey matter. In our case microscopical examination showed a congested condition of the vessels of the posterior horns.

Again, the fact that sensation was more affected on the left side than the right was interesting, as well as the different levels on the right side at which the touch, temperature, and painful sensations, were lost—observations which appear to support the view that different fibres and different tracts convey these sensations upwards to the brain.

[1] He has since successfully operated in a case of spinal tumour; but the details of it have not yet been published.

A CASE OF ATAXY WITH LOSS OF MUSCULAR SENSE.

BY BYROM BRAMWELL, M.D., F.R.C.P. (EDINBURGH).

In his very interesting and important communication on "The Muscular Sense, its Nature, and Cortical Localisation," which was published in the last number of this Journal, Dr. Bastian referred to two cases, recorded in abstract by Landry, in which the "muscular sense" was lost, while the tactile sensibility of the skin was very slightly, if at all, impaired.

The importance of cases of this description, as bearing upon the subject discussed by Dr. Bastian, is so obvious, that it need not be again insisted upon; while the rarity is shown by the statements of Dr. Ferrier: "that Dr. Bastian, with all his experience, is able to adduce only two cases, both hysterical, in support of his assertion, that muscular sense may be lost apart from affection of tactile sensibility;" and again: "I have elsewhere stated that defects in muscular sense are usually associated with defects in common or tactile sensibility, and that I am not aware of any case in which there has been loss or impairment of muscular sense without coincident impairment of tactile insensibility."[1]

In the following case of locomotor ataxia, the muscular sense is profoundly impaired, while the sensibility of the skin (except inasmuch as there is hyper-sensibility to cold and to pain) is practically normal.

Locomotor Ataxia of eight years' duration; extreme inco-ordination and total abolition of the Muscular Sense in the lower extremities; tactile sensibility of the skin of the lower extremities very slightly if at all, impaired; increased sensibility to cold, and increased sensibility to pain in the same parts. Dilatation of the thoracic aorta; aortic regurgitation, hypertrophy, and dilatation of the left ventricle of the heart.

The patient, O. C., a labourer, æt. 40, is an inmate of the Comley Bank Workhouse Hospital, Edinburgh, where, through the kindness of Dr. A. H. Peddie and Dr. Manners, I have had the opportunity of investigating and of studying the case.

[1] 'Brain,' April, 1887, pp. 91 and 92.

Previous History.—The patient states that his illness commenced some seven or eight years ago (in 1879 or 1880), with what he terms "weakness in the abdomen" and costiveness. This "weakness" gradually increased; his walking became difficult, and he suffered from sharp, shooting- (evidently lightning-) pains in the lower extremities.

During the year 1883, he was admitted to the Edinburgh Royal Infirmary, but he received no benefit; in fact, during his stay in hospital, the condition seems to have advanced rapidly, for he was able to walk into the Infirmary, but could not walk out. He has not been able to walk since.

On two or three occasions, at the commencement of his illness, he saw double; but he is unable to remember the exact dates.

He had syphilis sixteen years ago.

Present Condition.—The patient is pale and thin, and has a worn and somewhat haggard expression of countenance. He is quite unable to stand or walk, and only seems willing or able to move the lower extremities when he sees them. If, for instance, he is made to support himself on another man's back by means of his arms placed round the bearer's neck and with his feet touching the ground, he complains bitterly that he is going to fall, manifests great emotional excitement and appearance of fear; he says he does not know where his legs are. When the other man is made to walk forward, the patient makes no effort to move his legs, which are helplessly dragged and sprawled out behind. On one occasion he was strongly urged to try and make a step or two when in this position, with the result that one of his legs was thrown forward with great force, the motor discharge and resulting muscular contraction being out of all proportion to the movement which the patient desired to accomplish; in short, he seemed absolutely ignorant of the amount of muscular force required for the performance of the muscular act.

There is, however, no paralysis in the muscles of the lower extremities. The development of the muscles in the body generally, but more especially in the lower limbs (obviously here the result of disuse) is poor; the muscles are somewhat flabby, but the muscular power of all the muscles (tested for individual movements) is, considering their size, good.

When the patient is lying on his back in bed, he can execute any desired movement with the lower extremities, so long as he can see them; and the movements which he performs in this way are fairly steady and co-ordinate. He can, for example, perform with fair precision such movements as the following:—raising the foot high into the air and then bringing it slowly down, so that the heel rests on the point of the great toe or on the knee of the opposite (passive and extended) leg.

The power of co-ordinating the muscles of the lower extremities, when the eyes are closed, and the muscular sense in the lower extremities seem quite lost.

When the eyes are closed, and he is told to execute the movements described above, the leg is wildly thrown out, first in this

direction, then in that; and there is no appearance of any intelligent attempt to execute the desired movement, all knowledge of direction and all power of co-ordination being evidently wanting. After he has attempted to make a movement, such as the above, and the leg is allowed to remain in the position which it happens to have reached, the patient is absolutely and totally ignorant of its position. If told to take hold of the foot, he gropes about for it with his hands; it may be quite in the wrong place, often, in fact, far away from its actual position, and he only finds it by mere chance or by his hand coming accidentally in contact with some part of the limb, when he feels his way (guides his hand) down the limb to the foot for which he is seeking. When the lower extremities are placed in any given position (the eyes being of course closed while the passive movement is being made), the patient is also absolutely ignorant of the position in which the foot remains after the movement has been executed. He gropes about for the foot as before, and only finds it accidentally in the manner described above. He has, however, some knowledge of the direction in which a forcible passive movement of the hip or knee is being made. He can tell, for example, whether the hip is being flexed and extended on the abdomen, or whether it is being abducted or adducted, provided that the movement of flexion and extension or of adduction and abduction, are forcibly and rapidly repeated; but he is ignorant of the direction of the movement when it is slowly and quietly performed; and he is unable, even after quick and forcible passive movement, to tell the position in which the limb remains. In the same way he can tell when the knee is being forcibly flexed and extended. He does not, however, appear able to appreciate the direction of movements at the ankle joint, even when the movements are forcibly made and quickly and rhythmically repeated.

He is quite unable to appreciate ordinary difference in weight with the lower extremities. He does not, for instance, know the difference between the lightest and heaviest of the leather-covered balls with which I am in the habit of testing the power of appreciating weights, which weigh respectively three and twenty ounces.

He is, however, able to appreciate great resistance to muscular action. If, for example, he is told to make a powerful effort to raise his leg from the bed (the eyes being closed and the foot being firmly fixed down by the hand of the physician), he is conscious that some resistance is being opposed to the movement (possibly he acquires this information to some extent, at all events, from the respiratory movements associated with a powerful effort); but he does not seem to be aware that none of the desired movements has been executed.

There seems to be no loss of muscular sense in the upper extremities. He can (with precision and accuracy) touch the tip of the nose (the eyes being of course closed) with the fore-finger of either hand; and can, in the same way, bring the tips of the two fore-fingers in accurate contact in the middle line in front of

him—a delicate test of the patient's power of appreciating the position of the parts concerned and of his power of co-ordinating the necessary movements. He can at once tell the exact position of the upper extremities after passive movements; and can accurately describe the direction of any passive movement which the upper extremities are made to perform. He can also appreciate, with the upper extremities, slight differences in weight. He correctly differentiated all the test balls.

Sensory Functions.—The shooting-pains in the lower extremities are much less severe than they were a few years ago: they seldom now trouble him unless he is exposed to cold or draughts, to which he seems particularly sensitive. He often suffers from sharp shooting-pains in the abdomen (the exact locality he was not able very definitely to define). These pains came on in paroxysms; and are sometimes so severe as to make him cry out. The abdominal pains have never been associated with vomiting or other evidence of gastric disturbance.

The tactile sensibility of the skin of the lower extremities is practically unimpaired,—the patient can feel the touch of a soft and light object (such as a young tender hawthorn leaf) on any part of the thighs and legs and over most parts of the foot. He never failed to appreciate and locate correctly over any part of the lower extremities, including the feet, the touch of a hard object, such as the fine but blunt point of a tuning-fork.

(The tactile sensibility was not tested by means of the æsthesiometer, for I have found this instrument so unsatisfactory in uneducated persons and in dealing with diseased conditions, that I have practically discarded it as a reliable and useful aid to clinical investigation.)

Round the abdomen, at the level of the umbilicus, there was certainly a band of anæsthesia—measuring two inches in vertical extent; the loss of sensibility was by no means total, in fact it consisted rather of inability to locate accurately and correctly, and in delay in appreciating tactile impressions, than of abolition of tactile sensibility. This sensory impairment was more marked on the left than on the right side.

Painful impressions were vividly felt all over both lower extremities; there was, in short, well marked hyper-algesia. There was also some hyper-sensitiveness to impressions of cold, and perhaps slight loss of sensibility to heat, but this point was very doubtful.

Reflexes.—When the soles of the feet were tickled, the patient made some complaint of pain and drew up the legs.

The knee-jerk on each side, and the plantar, cremasteric, and abdominal reflexes were entirely absent.

Urination was said to be attended with considerable difficulty. Calls to urinate come on with great suddenness, and have to be immediately obeyed (*precipitant urination* from loss of inhibitory control); much straining is required to empty bladder; and this, he says "can only be effected when he curls himself up and bends forwards," in other words, when he places himself in such a

A CASE IN WHICH PARALYSIS OF THE SPHINCTERS AND INCONTINENCE OF URINE WERE, TOGETHER WITH TORPID INTELLECT, THE CHIEF SYMPTOMS OF SYMMETRICAL DISEASE OF THE CORPORA STRIATA.

BY JONATHAN HUTCHINSON, F.R.S., LL.D.

Mr. S., the manager of a bank, aged 54, but looking younger, consulted me, at my own house, on account of incontinence of urine, on January 30th, 1885. The incontinence had occurred almost solely in the daytime. He said that when the desire came he could not wait, and that the urine escaped before he could get to the utensil. Yet there was no urgency of desire. Sometimes the escape would be almost without warning. This trouble had begun six weeks before, and his accidents now happened two or three times every day. I thought, of course, of enlarged prostate, but on passing a catheter found only a small quantity in the bladder, and on using the finger per anum the prostate was felt to be but slightly enlarged. In making the latter examination I realised that the sphincter ani was quite relaxed. It allowed the finger to slip in and out without gripping it in the least. The lower bowel was loaded. He denied that he had experienced incontinence of fæces, but for this the very solid condition of the fæces probably accounted. I now thought of the spine; but on enquiring, he denied that he had any weakness or alteration of sensation in his legs. He had had no headache, and asserted that his incontinence of urine was his only symptom. On looking at his pupils, I found that their margins were extensively adherent in both eyes. This much interfered with examination of their activity. He said that he had usually been a bad sleeper, but was now sleeping better. He looked well, seemed inclined to make light of his ailments, and on this occasion I saw no reason to suspect that he had any cerebral disease. I thought that he was about to become the subject of some form of spinal paralysis. On the following day I received a letter from his brother-in-

position as to bring the full purchase of the contracting abdominal muscles to act upon the bladder.

The *bowels* are obstinately constipated.

The *pupils* are small, the right measuring 2 and the left 2½ mm.: it is doubtful if the pupils contract to light, if there is any contraction it is certainly very feeble; contraction associated with efforts, fixation for near objects and accommodation is active.

Sight, both for white and colours, seems good, and the other casual senses are normal.

Powerful pulsation can be felt in the supra-sternal notch, and there is distinct impairment of the percussion note over the manubrium-sterni. A well-marked double aortic murmur is audible over the course of the aorta and up and down the sternum. The pulse is typical of aortic regurgitation. The left ventricle is moderately enlarged, hypertrophied and dilated. The appetite is good, the tongue is clean, and the other systems and organs seem natural.

of his feet had been a little numb, but they had not become more so of late. By another examination I confirmed the observation that his sphincter ani was quite lax. He repeated that he was sleeping better than he had ever done, and alleged that his head and mind were all right.

It was now arranged that he should leave town, and he went to Scotland with his wife. Here he became rapidly worse in all respects, and after three weeks they returned.

In the middle of March I saw him at his own house. He had lost flesh and strength, and looked more pale, though not markedly ill. He walked into his drawing-room to meet me, and greeted me cheerfully; but he did not seem to know what to do, and I had to ask him to sit down. He conversed about his journey, but in a vague and uninterested manner. I found that he did not know the day of the week, but after I had told him, he corrected me as to the day of the month. All other symptoms were as before. His wife said that she was obliged to make him get out of bed three or four times in the night, or he would certainly pass water without caring to rise. Once when on a visit to a friend, he had passed water in the room, and seemed quite careless about it. I made him whistle, and ascertained that he had no paralysis of the seventh nerve, though it was perhaps feeble. Within a week of this visit Mr. S. died. I had not seen him again, but I obtained the following facts from his surgeon and his wife. During the week he got a little worse, but he had seemed decidedly better on the day preceding his death. He had been up every day, and about the house as usual. At length, one morning after a night of unusually heavy sleep, he had been difficult to wake, and when roused and asked if he would get up, had replied distinctly, "No." His wife kissed him, and he kissed her back, but then immediately sank into sleep again. The sleep became more profound, and they could not rouse him. His left arm and his right leg were engaged in twitching convulsive movements. The sleep passed into coma, which was attended by stertor. Then the respirations became intermittent, and finally they ceased. He died six hours after he fell asleep.

We got permission to make a post-mortem. It was done, under considerable difficulties, about thirty-six hours after death. In the inner side of the left corpus striatum was a soft pink semi-gelatinous growth. This growth bulged into the ventricle, and was quite smooth on its inner surface. On its outer side it merged without any boundary line into the substance of the corpus. The thickness of the new growth was about half an inch. A little to its outer side, in the white substance of the hemisphere, were two small islands of

law, which showed me another phase of the case. From it I make the following extracts :—

"After our interview with you yesterday I went to Beckenham and saw my sister, and found her in great trouble about the condition into which Mr. S. seems to be rapidly falling. It appears that he *has* occasionally—certainly three times—had involuntary motions of the bowels. But her chief anxiety arises from the utter mental prostration, and physical inertia which seem to be overtaking him. This inertia is so great that he seems to be incapable of making the slightest effort. For instance, he not only cannot exert himself to get in time for his daily train, but with plenty of time to spare to catch the next, he will lose that also. And it does not seem to trouble him that this should happen frequently. Then when he comes home he will stand in an aimless way with coat and gloves on for a long time, not seeming to know or to care what he is going to do next. Mrs. S. had the greatest difficulty to get him off in time for his appointment with you. For nearly two hours, while he was dressing and getting his breakfast, she hardly dared to leave him, and it was only by having a cab waiting at the door—although the station is only five minutes' walk—that he was got off in time. Another little thing she notices is that he will sometimes grasp her hand or arm sharply enough to cause pain, or will 'flick' one of the boys with anything he is carrying, acting apparently without thought, and from some momentary impulse. In short, he is not himself at all."

A few days later, one of his colleagues in his place of business called on me and gave a similar report. He said that Mr. S., from having been an austere and precise man, had become the reverse, that he was now effusive and jocular in a manner which was quite contrary to his wont. Above all, that he was constantly passing his water as he sat in his office-chair without seeming materially annoyed about it.

On Feb. 7, a week later, I saw Mr. S. again, and ascertained the following points: that his knee-jump was good, that his pupils acted well, and that he could stand well either on his heels or on tip-toe. He told me that he had not lost sexual power, but had always been very moderate. He admitted, on being pressed, that he had occasionally a peculiar feeling in his head, which he could not explain. It was akin to giddiness, but he was not giddy. His memory and intelligence appeared to me after a long interview to be perfect, but he seemed rather too cheerful and smiled too much. It should be said that there had been no stage of elation such as occurs in most cases of the general paralysis of the insane. His sight was good, and he had no morbid changes in the fundus. He said that for ten or twelve years past the soles

similar structure, as large as cherry-stones, but quite ill-defined. Neither these nor the larger growth had any softening or congestion around them. At this level the opposite hemisphere appeared healthy, but on slicing it more superficially, there was found just above the corpus, or in its upper part, a large softened area broken down by blood clot, but having, at one part, an indistinct lining membrane. A clot the size of a walnut, soft and jelly-like, extended from this patch of disease into the ventricle.

There was not a trace of disease at the base of the brain, nor in the pons or medulla. The arachnoid over the surface of the hemispheres on both sides was adherent, but there was nothing very unusual or which appeared to be recent.

The examination of the brain was not quite so precise as could have been wished, but it was done by candle-light, and under disadvantages. It may be taken as established that there was destructive disease of the anterior and inner parts of the corpus striatum on both sides, and that no inflammatory mischief had been set up. The hæmorrhage was on the right side only. No doubt this bleeding afforded the explanation of the suddenly fatal event.

My eldest son made a careful microscopic examination of the tumour, and has preserved sections. I append his report.

"After hardening with the usual reagents, some satisfactory sections were obtained, fully bearing out the diagnosis of sarcoma made at the post-mortem examination. Those parts free from blood-infiltration showed spindle and round cells closely massed together, and tending to be arranged somewhat in lines. From the size and shape of the cells, the tumour should perhaps be regarded rather as a mixed round and spindle-celled sarcoma, than as a typical glioma, though presumably its origin was from the neuroglia.

"Especially in the neighbourhood of the hæmorrhage some vessels of fair size were seen, with a considerable number of capillaries, probably of new formation, in which the thick cellular wall almost concealed the lumen. Even in some parts which to the naked eye looked like blood-clot, tumour elements could here and there be made out."

It is certainly remarkable that a lesion in this position should have been attended by so much mental disorder and by no limb paralysis. Sphincter-paralysis had been the most conspicuous symptom throughout, but his limbs, so far as he would admit, had never failed. He could always grasp firmly with his hands, and could walk. His walking power had, however, certainly diminished. It is very possible that the symmetry of the disease had been productive of bilateral weakness, and that thus the defect had been less easy of discovery.

After his death, his wife told me that she had sometimes thought that his right limbs were weak, but one of his sons who had been much with him had formed the same impression as to his left. It was certain, they said, that in walking he sometimes reeled a little to one or the other side.

Dr. Bright, in the second volume of his "Medical Reports," has given a portrait, which shows a closely similar condition of the corpus striatum. The chief differences indeed, between his case and mine consists in the fact, that in his the lesion was on one side only, whilst in mine it was symmetrical. If we keep this fact in mind, we shall find that the symptoms in the two cases were nearly parallel. In Dr. Bright's case, as in mine, incontinence of urine was an early symptom, and throughout one of the most conspicuous. His patient was a man of 40, who had been a soldier. The earliest symptoms observed had been that he would occasionally fall in the street, and lose consciousness for a moment or two. He complained of pain in the forehead, and had occasional absence of mind with slight weakness of the left side. Next he began to constantly pass urine in bed, and occasionally fæces also. After this there was general muscular and mental debility, with strabismus and diplopia. "His manner was peculiar, and his conversation incoherent; he stood for some minutes together in one position as if lost in abstraction; when spoken to, he answered a question with some consideration apparently, but then at once ran on to something else, without, however, saying much, but making some statement perfectly without foundation." The symptoms, including the premonitory group, had lasted a year when Dr. Bright first saw him, and he lived, afterwards, in Guy's Hospital, for six months longer. He had no facial paralysis, nor, so far as the notes inform us, any defect of sight or hearing. A few days before death it is noted: "His general manner and aspect quite unchanged. He stands about musing, and if you ask him how he finds himself, answers after a few seconds of consideration, 'I am not quite dead.'" He had gradually become more dull and heavy. It was only during the last week that his imbecility and debility were such that he could not leave his bed. He had on the last day great difficulty in swallowing.

Dr. Bright does not describe the condition of the corpus striatum as implying new growth, but from the appearances delineated, it probably did so. He says that two-thirds of corpus were "involved in softening, and presented a filamentary and watery-brown appearance." The rest of the brain-substance was healthy, but over a space two inches square on the right hemisphere all the membranes adhered firmly. There was some general serous effusion. In commenting on the case,

Dr. Bright states: "The appearances after death were confined to softening of the corpus striatum on one side . . . but it had not the precise appearance of an apoplectic clot."

In Auvert's magnificent collection of pathological plates there is one (Pl. 68, Fig. I.) which shows the section of a brain in which appearances are represented somewhat similar to those I have described. The tumour involved both thalamus opticus and corpus striatum. The patient, a man of 56, had died after a week's illness, following a sudden seizure during anger. He had been liable to hemicrania. The disease is described as red softening, and not as new growth.

CASE OF THOMSEN'S DISEASE.[1]

BY FRENCH BANHAM, M.A., M.D.

Physician to the Royal Berkshire Hospital.

THE patient, a brief report of whose case is subjoined, came under my notice a year and a half since. I am now again in communication with him, and as the disease from which he suffers is one of great rarity, I feel that so good an example of it should not pass unrecorded.

The affection of which he is the subject is known as Thomsen's Disease (so named from the physician who first described it, and who is himself a sufferer from it), and which consists almost wholly in spasms of the voluntary muscles coming on at the outset, or upon any change of motion, and quickly passing off again. Few, if any cases, have been recorded in our English journals, so in pointing out any special peculiarities exhibited by my patient, I take as my standard of comparison the descriptions of the disease as detailed in some of our best treatises on Diseases of the Nervous System. The affection is generally spoken of as being hereditary; but in this case it seems not to be so, as no other member of the family, as far as can be ascertained, has been similarly affected, nor is there any tendency to disorders of the nervous system, and the patient is one of seven, the eldest being 22, and the youngest 9 years of age. Further, it is said to be congenital, some indications of it having been observed in the cradle; but my patient, now 18 years of age, had no symptoms until he was 12, and these did not become severe until two years ago, when he was taking swimming lessons, and for forty consecutive days was accustomed to remain in a cold bath for upwards of an hour together. He is an intelligent youth, well-developed, especially as regards the legs, and muscularly strong, while his general health is good.

The first time he noticed a spasm was, as I have said, six years ago. He was jumping over a flower-bed in his schoolmaster's garden when a spasm of the legs occurred; he could not lift his feet so as to clear the flowers, and his master

[1] Read before the Reading Pathological Society, Dec. 8th, 1886.

caned him for the damage done. He remembers the increasing frequency of the symptoms from that time, marked by his inability to join in such games as "Hare and Hounds," as he had formerly done.

The symptoms vary very greatly at different times. When severe, a spasm comes on as soon as he has altered the condition of a muscle after a period of rest. Thus, after having sat down for a little time, when he rises to his feet, he cannot stir his legs because the spasm immediately occurs, and for the same reason he cannot balance himself unless he has gained his equilibrium before the spasm is developed. This leads to his being thrown down in trains and tramcars on attempting to stand up after sitting, unless he is able first to grasp the rail. Again, after getting into a conveyance and sitting down, he cannot bring his heels towards the seat at once, but often kicks the opposite passenger through his legs sticking out stiffly; and as an illustration of the transient character of some of the spasms, I will quote his own remark—"that he generally gets his legs down before the fellow he has kicked begins to swear." The spasms are also very marked in his attempt to walk upstairs. He has very great difficulty in lifting each leg on to the first and second steps respectively. A slightly less difficulty in the next two series of motions, and a continually decreasing difficulty in each successive series, until he has mounted, say, twelve or fourteen steps. After this he has no further trouble for the time being. In such circumstances, twelve or fourteen seconds is the longest time that the difficulty lasts. Again, on bending the arm sharply at the elbow, the hand may readily approach the shoulder, and will return about half-way back, when a spasm occurs which holds it there for a few seconds. Again, on opening the mouth, it is fixed for a few moments before he can close it. The difficulty vanishes, however, after a little exercise, and will not recur unless brought on by fright or great excitement. His arm or jaw seems never to remain rigid for longer than about three seconds; the time, however, varies from this to a momentary check in the movements. When a muscle has become stiff, as he expresses it, he does not suddenly acquire full power over it again, but in a longer or shorter time he can begin to move the limb, slowly at first, but with increasing vigour as time passes.

The disorder varies in intensity, and when less severe, the spasms do not take place so immediately at the outset, but only after a series of motions. For example—he will mount nine steps readily when a short spasm will occur, lasting perhaps until he has reached the twelfth, and necessitating his grasping the banister to prevent himself falling.

There are certain circumstances and states of mind which seem to influence the spasms. They are slightly more marked, and last longer when he directs his attention to them; but his will does not seem to affect them at all. Under fear or excitement, the symptoms will suddenly appear at other times than at the outset of voluntary motion. As an illustration, I may mention that when walking one day with a friend in a railway tunnel, and being surprised by the near approach of a train, the spasms suddenly set in in the legs, so that his companion had actually to drag him out of the train's way. And again, sometimes on getting into a train he has literally to fall into the carriage if he is greatly excited or the train is on the point of starting. In the case of the hands, the spasms so affect them that he is frequently unable at once to let go objects that he has grasped. The muscles of the tongue and larynx, so far as I can ascertain, have never been affected. He seems always to have led an active life, so that I am unable to say what the effect of rest would be upon the tendency to spasm. It seems, however, that when after remaining in one position subsequently to taking prolonged exercise, he recommences movement, the spasms at the outset occur very promptly and strongly. The disease is considerably influenced by cold, rainy weather, and he tells me that he generally knows when it is going to rain by the strength and character of the spasms. The tendon-reflexes are scarcely so strong as is usual in health, and the left patellar tendon-reflex is rather weaker than the right. In regard to the superficial reflexes, tickling of the soles of the feet elicited no response.

The electrical reactions of the muscles are not precisely coincident with what has been recorded in other cases. Momentary stimulation of the nerves did not cause more than a momentary contraction of the muscles either in the case of the opening or closing contractions of either pole, or in the case of a momentary stimulation with a rapidly intermittent current. The contractions produced on starting a current through a motor point were momentary and normal, although the current was maintained constant after making. I mention this because it has been stated that in some cases in certain muscles an unbroken current has caused peculiar wave-like contractions, about one per second passing from the negative to the positive pole. An intermittent direct current, however, that is an intermittent current of uniform direction, maintained a constant contraction of the muscle with either pole. In the case of one muscle only, the vastus internus, peculiar contractions and relaxations were observed during the application of the intermittent current, the active electrode being

placed over the motor point of the muscle. At first a normal contraction lasting about two seconds occurred, and then an irregular recurring series of contractions and relaxations. In all the experiments the sequence of the polar reactions was found to be normal, with the slight exception that the ACC and the AOC were produced together. The first contraction was produced with a kathodal current of 6 milliampères, the diameter of the electrode being 1 inch, the ACC and the AOC with a current of 8 milliampères, although the opening contracture was more vigorous than that produced by the closing of the anodal current with the same current strength. The KOC did not occur until the current had been increased to 10 milliampères.

I have not made any microscopical examination of the muscular fibre; but in a case observed by Erb many fibres were seen exceeding the normal maximum in size.

I must thank Mr. J. Lester Taylor, of Sheffield, for the kind assistance he has given me in working out some of the details of this case.

I.—A CASE OF RECURRING ATTACKS OF TRANSIENT APHASIA AND RIGHT HEMIPLEGIA.

II.—A CASE OF TUMOUR OF THE PINEAL GLAND.

BY EDWARD O. DALY, M.A., M.D. (OXON.), M.R.C.P.,

Physician to the Hull Royal Infirmary.

A GENTLEMAN, aged 68, was suddenly seized, on March 11th, at 6 A.M., with aphasia and right hemiplegia. The attack lasted about five minutes, and passed off as rapidly as it came on. He had suffered occasionally for years from gout, most frequently affecting his bladder. The first sound of his heart was found to be feeble; his arteries were thickened, and his urine was pale with a well-marked trace of albumen, and a sp. gr. of 1010. His temperature was normal; his pulse 80. On March 13th he had two attacks of a precisely similar kind to the first, each lasting about half an hour.

March 14th there were ten attacks, the shortest lasting about ten minutes, the longest almost an hour.

March 15th there was one attack of ten minutes' duration, and three in succession, with about a ten minutes' interval between, each extending over three hours.

Since March 15th he has been quite free from an attack of any kind.

I saw this gentleman during several of these attacks. They came on perfectly suddenly without any premonitory symptoms, and passed off as rapidly. He would suddenly say, "I am all right again."

There was never any kind of convulsion.

During the attack the intelligence was certainly affected, but it was difficult to estimate to what extent.

When asked to put his tongue out he did so at once, and also closed his eyes, and performed intelligently all actions asked of him. The aphasia was not complete, but the words he had at his command were limited, and frequently misapplied; and in some of the attacks he was quite unintelligible.

The hemiplegia was also partial, more marked in the arm than the leg. He could move the right hand to a limited

extent, and was able to grasp my hand feebly. There was also slight anæsthesia of the right side.

The attacks in every instance ended abruptly, the speech becoming instantaneously perfect. The power in his right side returned almost as rapidly, but after the longer attacks a few minutes elapsed after the return of speech before he could use his arm and leg perfectly.

In the intervals between the attacks he felt quite well, with the exception of slight frontal headache.

The only treatment consisted of perfect rest, a few grains of calomel, and a mixture containing small doses of iodide of potassium and citrate of lithia.

When I first saw this case I was inclined to think it due to transient thrombosis of the left sylvian artery. On referring to various books on the nervous system I find Dr. Bastian, in his book 'Paralysis from Brain Disease,' mentions spasm of the vessels as one of the causes capable of rapidly producing a hemiplegic condition. He remarks unfortunately there is a great deal of uncertainty on this point.

As these attacks came on and passed off so rapidly, the speech and movements of the right side in the interval between the attacks being perfect, the case seems more readily explainable as due to spasm of the sylvian artery, than to thrombosis or any other cause. The case, at any rate, seems a very unusual one.

In some cases polyphagia has been ascribed to pressure of the tumour on the "diabetic centre," but no such explanation is admissible in this case, the urine being normal throughout.

In other cases it depends on the implication of the cerebral centre of the stomachic branches of the vagus.

Again, unnaturally large appetites have been observed in disease of the post-cerebral lobes, which must have been subjected to abnormal pressure in the present case. It may be that this symptom has been noted in other cases of tumour of the pineal gland, though I am not aware of it.

This gland, now shown to be a rudimentary "parietal eye," was at one time believed to be the seat of the soul, and had the patient lived and died in those days, his moral and physical deterioration would doubtless have been brought forward as strong evidence in favour of the correctness of this theory.

Case II.—W. O., single, a labourer, aged 23, was admitted into the infirmary on October 30th, 1885. There was no family history of consumption, nor of any other constitutional disease. He had not had syphilis, and there was no history of any former head injury. He was strong and healthy until two or three weeks prior to admission, when he believed he had been

struck by lightning, and had complained since that time of more or less constant headache and photophobia. On admission he was found to be a strongly-built man, his gait was peculiar, and resembled the walk of a drunken man, swinging his trunk from side to side and from back to front. His gait was worse when he closed his eyes. As he lay in bed, he could move his legs freely in any direction. His arms were not affected, and the patellar tendon reflex was normal.

The patient complained of pain at the back of his head. His vision was impaired, pupils dilated and equal, contracting but slightly to light or accommodation. An ophthalmoscopic examination revealed double optic neuritis with swollen disc. His other senses were normal. He was constantly sick, irrespectively of food. His urine was free from albumen and sugar, and not increased in amount. Nov. 11th, slight paralysis of the left external rectus was noticed, and on Nov. 20th facial paralysis on the right side. Nov. 25th, there was some difficulty in articulation for some hours, which passed off entirely.

During the latter part of November and December attacks of spasmodic contractions of the muscles of the limbs and trunk occurred from time to time. At times his mind seemed much affected: he would get out of bed several times during the night, and occasionally wanted to fight. From the beginning of January, 1886, he began to develop a very large appetite, and his vomiting considerably abated.

He remained *in statu quo* for some weeks, and was discharged, at his request, January 29th, 1886. Re-admitted February 27th, 1886.

It was now noticed for the first time that the hearing of both ears was defective, and he was found to be almost blind. He was said to have had occasional fits. He could only walk and stand with assistance, though he could move his arms and legs freely. There was no anæsthesia. His appetite was soon found to be enormous, and the patients were in the habit of giving him what they left on their plates. On one occasion eight or nine patients put their pudding at his disposal, in order to see how much he would take; all this he finished without trouble. During the time he was in the infirmary he put on nearly five stone. The urine remained normal in all respects. During March and April he seemed to lose all intelligence, became almost completely deaf, and his sight failure passed into total blindness. He also completely lost control over the bladder and rectum. When pinched or moved he would give a kind of grunt, but hardly ever uttered a sound, except when he wanted food. He was occasionally observed to lie with his head drawn back, which was found to depend on a tonic contraction of the muscles of

the back of the neck. He had from time to time epileptiform convulsions, in which the movements were said by the nurse to have been general. I never however myself saw him in one of these attacks after his re-admission.

During the last week or ten days of his life his pulse and respiration became very rapid, and he passed into a state of coma several days before his death, on May 19th, 1886.

At the post-mortem examination a circumscribed tumour was discovered extending backwards from the point which corresponded to the situation of the pineal gland. It was loosely attached to the upper surface of the cerebellum, lying between it and the under surface of the cerebrum. In its growth it had seriously pressed upon the neighbouring structures, especially the corpora quadrigemina, which were soft and flattened. The tumour was two inches in length, one and three-quarters in breadth, and half an inch in thickness; and is well shown in the photograph kindly taken for me by Mr. Howlett. Subsequent microscopic examination showed the tumour to be one of alveolar cancer: this view of its minute structure was corroborated by Mr. Young, of Manchester.

Remarks.—The case was diagnosed with considerable confidence as a tumour of the middle lobe of the cerebellum. The attacks of spasm observed have been usually ascribed to tumours in that situation, and this growth no doubt must have pressed upon the middle lobe. I have lately however seen a case in which rigidity and tetanus-like seizures were absent from first to last, but in which at the post-mortem the middle lobe was almost entirely destroyed by a soft growth. I am therefore inclined to agree with the explanation put forward by Dr. Sharkey in his Gulstonian lectures last year; namely, that the symptoms in this case depend, not upon the pressure on the middle lobe of the cerebellum, but upon the fact, that the tumour was in such a position and of such a size as to cause pressure on the pons and medulla.

The great impairment of vision present at such an early stage of the disease was doubtless due to the fact, that the tumour from the first pressed upon the corpora quadrigemina.

Although the tumour was diagnosed during life as cerebellar, there was one symptom, polyphagia, which is not, I think, characteristic of tumour in that situation. That central disease may give rise to ravenous appetite is frequently well exemplified in certain forms of insanity. When it occurs in the case of cerebral tumours it does not, as a rule, prevent the progressive emaciation which is so commonly observed towards the close of such cases. In the present case however the patient put on weight up to the last, and was five stone heavier at his death than on his admission.

TWO CASES OF PECULIAR MOVEMENTS IN CHILDREN.

BY W. HALE WHITE, M.D.,

Senior Assistant Physician to Guy's Hospital.

CASE I.—A. A., æt. 14, came to my Out-patients' department on March 2nd, 1886. The notes made at the time are as follows:—

He is healthy-looking and very intelligent, being at the top standard but one at the Board School. He is an only child; a paternal uncle is in an asylum, otherwise the family history is very good. Forceps were used at birth, from which time his mother has noticed irregular twitchings of his muscles. When he was three months old, she took him to the Great Ormond Street Hospital. The doctor said he would grow out of it, and gave him cod-liver oil; but he has not improved.

Present Condition.—On watching the boy, occasional slow involuntary movements of the fingers and forearms are seen more on the right side than the left, where indeed they are hardly perceptible. These movements affect the face and both extremities on the slightest attempt at voluntary movement, and indeed they may at times be seen on the face without any voluntary motion. They are least marked in the lower extremity. When at their greatest they are extreme, the platysma and trapezius showing the irregular spasmodic contractions very well. It cannot be made out that the muscles supplied by the 3rd or 5th nerves are affected. Perhaps the action which shows the trouble to the greatest perfection is unbuttoning his shirt: he makes the most irregular and violent attempts with his right hand; the right side of the face is distorted; the left hand tries to help, but is affected in the same way but to a less degree, and finally his mother has to help him; but if he employ only the left hand he can after some time manage it. His handwriting is hardly legible, and he has to hold the right hand, which holds the pen, steady with the left. Owing to the face-affection the speech is irregular and jerky. The abdominal and thoracic muscles are not affected, but the affection of the right leg causes an irregular gait. All his troubles are worse when his attention is directed to them. The knee-jerks are normal, as is the triceps on the left side; but the attempt to get it on the right sets up the irregular movements. There is ankle-clonus on both sides, and diminished superficial reflexes all over the body. No paralysis or contraction, no atrophy, no paradoxical contraction. Electrical reactions all normal. No disturbances of

sensation. Ophthalmoscopic and laryngeal examination showed normal results. Special senses normal; no trophic changes. No asymmetry of head, limbs, &c.; no wasting; throat normal; uvule bifid; dentition is said to have been delayed. There is a narrow patch on the right leg.

March 1887.—For the past year he has been treated with iodide of potassium and tonics, but without any benefit. A most thorough examination showed the condition to be unchanged, except that no ankle-clonus could be obtained, and the reflexes were not so lively. He has not suffered from headache or sickness. As he has got used to me he does not make quite such extreme movements as he did at first when talking to me, but he is just as bad as ever with strangers. This patient was exhibited at a Clinical Meeting of the Neurological Society last March.

Case II.—C. L., æt. 10, admitted under me into Philip Ward, Feb. 26th, 1887, for spasmodic movements of the right arm and right facial paralysis.

Family History.—The maternal great-grandfather had fits; he died, æt. 60, paralysed. A maternal great-aunt had fits; a first cousin (mother's sister's child) has fits and is weak mentally. The patient is the eldest of four. The second, a boy, was born with a large head and is very excitable. The third, a boy, has often been operated on for nævi. The fourth, a girl, is healthy. The mother is healthy, but her mother from childhood has had tremblings of the head and right arm: she has had thirteen or fourteen children, and is now almost bedridden.

Personal History.—His birth was a "hard confinement;" he was a nervous child, easily frightened, screaming when the clock struck. In May, 1886, he woke up one morning feeling sick and feverish. He was kept in bed a week, as he had a sore throat; his mother noticed he was a little shaky, both hands being tremulous. He quite recovered. In July, 1886, his mother again noticed tremulousness, confined to the right hand, though he could still feed himself; but by October it had got so bad that he could not write. At this time he described a pain which began in the right middle finger and extended up the arm. He was now removed from school, where he had been looked upon as a dull boy, although he was in the second standard. His speech was noticed to be affected. By November the right arm was useless. He was taken to a doctor, who drew attention to slight facial paralysis and dragging of the right leg, and said he had either chorea or paralysis. He was taken to a hospital, where the same diagnosis was made. Change of air was frequently tried, but he got no better, and was admitted into Guy's. No rheumatism.

Condition on Admission.—Well-nourished, healthy-looking, dark hair and eyes; sits up in bed, amusing himself with toys and using the left hand exclusively. Has a small nævoid patch on the left outer malleolus. There is nothing noteworthy about the mental condition. As far as the facial paralysis permits, the pantomimic speech is good, as is the spoken, except for slow drawling but

distinct utterance; this also would appear to be due to his paralysis. There is no aphasia. He cannot write. The only defects of the eyes are that the left pupil reacts better than the right; also, on looking up, the right eye is turned further up than the left; and in moving up, it performs slight horizontal oscillations. Hearing, taste, and smell normal.

Motor Functions.—There is paralysis of the muscles supplied by the right facial nerve, but it is very variable, the mouth being drawn violently to the left at one time, but not a few minutes after; both orbiculares palpebrarum and occipito-frontales muscles act equally and well. There is some paralysis of the left levator palpebræ. The tongue is protruded normally. The uvula and palatine arches are equal. The power in both arms is diminished, but especially in the right. There is no paresis of the legs. When he sits naturally, the following is the position of the right upper extremity. The humerus is at the side, the forearm at a right angle over the chest, the hand is clenched, the thumb being adducted and pressed into the palm by the fingers. At irregular intervals spasmodic twitchings come on, mostly affecting the elbow joint, the closed hand being thus rubbed up and down the sternum and making an excursion of about two inches. About a dozen of these twitches occur in as many seconds, and then the arm is still again for some minutes. If he is being watched, the movements are continuous; and if he is told to take one's hand, the whole extremity is irregularly thrown about before the object is accomplished. All this time the hand continues flexed, and he has to open it with the right before he can grasp anything. Occasionally the hand will open spontaneously. Although there is generally much rigidity at the elbow and hand, still if they are opened with some force they soon become flaccid, and also they may at times be opened with less trouble than at others; but the flaccidity soon gives way to contraction again, and the hand shuts up. The left arm is normal, except that at first there appeared to be some slight choreiform movements of it; but after he had been in a day or two, they disappeared. No wasting, anæsthesia, or reflexes of arm. In the right lower extremity the knee-jerk is absent, as is ankle-clonus and paradoxical contraction. As he lies in bed the foot is persistently extended at the ankle, the sole is arched, the toes are extended at the metatarso-phalangeal joint and flexed at the phalangeal; plantar reflex very sluggish. On walking, this rigidity of the foot passes off, but the patient has some inco-ordination, the right leg being swung too far round, and he very quickly throws the weight on to the left. Right lower normal. No electrical examination permitted.

Cases like these in some particulars have been recorded by Ross[1] and by Hadden.[2] Most of the cases by Ross had more spastic rigidity than either of mine; the last is perhaps

[1] 'Brain,' Vol. V. p. 344.
[2] 'Clin. Soc. Trans.,' vol. xviii. p. 221.

doubtfully congenital, although it may be looked upon as a congenital disease supervening late in childhood; the first certainly is congenital. It is evident, on reading the accounts of them, that there is great difficulty in assigning to any one lesion the production of the symptoms in either case.

Until we know whether a lesion is present, and if so, what its position is, we have hardly enough basis for speculation as to causation of the symptoms. I intend to keep an eye on these two patients, and, if I outlive them, to complete the account of them. The cases that have been published of congenital brain deficiency in children producing symptoms, differ so among themselves, that they hardly form any guide in other cases. It is only by the recording of all these vague cases in which a diagnosis cannot be made that we can hope to eventually classify them, when we have had the opportunity of comparing a large number.

MIGRAINE ATTACKS FOLLOWED BY TEMPORARY PARALYSIS OF THE THIRD NERVE.

BY C. W. SUCKLING, M.D. (LOND.), M.R.C.P.,

Physician to the Queen's Hospital, Birmingham.

R. R., a youth aged 18, a tool maker, applied at the Birmingham Eye Hospital for treatment for dropping of the left eyelid. Dr. White found that there was complete paralysis of the left third nerve, and there being a history of previous similar attacks, he kindly sent the case, as one of interest, to me.

Since infancy the patient has been subject to severe headaches, occurring at varying intervals. During the last four years the attacks have occurred more frequently and have been more severe, and for the past two years he has had an attack pretty regularly every fortnight.

The patient's aunt used to suffer severely from periodical headache; but neither of his parents have been affected by this or any other neurosis.

An attack lasts two days, and takes the following course:—He first feels a slight pain above the left brow, which gradually increases in intensity, and is followed by a flow of saliva into the mouth, and he feels very cold. He at once goes to bed on feeling the onset of an attack, but cannot sleep, and he has to remain in bed for two days without being able to sleep or to take any food.

The pain increases gradually, and attains its maximum in twenty-four hours: it then as gradually subsides, and ends completely in another twenty-four hours.

The pain remains localised over the left brow; he does not vomit, and there are no ocular spectra or paræsthesiæ of any kind, except that he complains of a nasty taste in the mouth.

After an attack his scalp is tender, and he cannot comb his hair.

The left eyelid was first noticed to be drooped after an attack in his infancy, and this has occurred very frequently since.

Three years ago, after a severe attack, his left eye was completely closed, and the eyeball turned outward.

The eye is not affected in every attack, but only in the severe ones; and the more severe the pain, the greater is the amount of dropping of the lid which follows.

The left eyelid begins to drop at the end of an attack when the pain is wearing off. The lid takes twenty-four hours to drop completely, and in another twenty-four hours has usually recovered. The eye is not always turned outwards, but it has been so on two separate occasions, and then the paralysis lasts longer, not recovering for a week after the attack.

His mother attributes the increase in the severity and frequency of the attacks to his having attended a night school during the past two years.

On examination a week after an attack the left pupil was a little larger than the right, and responded normally to light and to accommodative efforts. The left palpebral fissure was a little smaller than the right, and the upward movement of the left eye was defective, there being diplopia in looking upwards. He complains that the sight of the left eye is not so good as that of the right. Dr. White reports that vision is normal, and that when he saw the boy the paralysis was complete both of the internal and external muscles of the eye.

The salivation that occurs in this case during the attack is also of interest.

In another case of migraine at present under my care a profuse clear discharge runs from the left nostril during the attack—the pain being located over the left brow; and in this case the scalp is tender, and painful lumps are occasionally present on it.

Migraine in some cases is so serious a malady that it completely incapacitates its victims for their duties, and in the majority of cases is a most serious drawback. Its victims can usually tell what are the exciting causes, and affirm that fatigue, worry, reading, cold winds, damp, wines, pastry, &c., will bring on an attack; but however careful they may be, they cannot rid themselves of their burden. Cigar-smoking is a very powerful exciting cause of an attack. A medical friend of mine invariably suffers from a severe attack if he smokes cigars.

In the above-mentioned case guarana in thirty-grain doses, taken every hour at the commencement of the attack, has given great relief. The duration of the attack is shortened to a few hours, and though the patient has had attacks, they have not again been followed by the paralysis.

The only other case of migraine in which the third nerve has been paralysed that I can find recorded is that published by Dr. Saundby in the 'Lancet' of January 10th, 1885.

Critical Digests.

RECENT OBSERVATIONS ON PROGRESSIVE MUSCULAR ATROPHY.

BY H. H. TOOTH, M.D.

THE great impulse given to the study of the disease, or rather group of diseases, known as Progressive Muscular Atrophy, by the researches of Erb, Charcot, Landouzy and Déjérine, Schultze and others, renders a review of the subject a desideratum. Such a review to date appeared in 1885, from the able pen of M. Charcot, but since then further advances have been made. The object of the present article is rather to place before the reader the most recent views of authors on the subject, than to criticise those views. It will be seen that recent anatomical observations tend rather to upset our preconceived ideas as to the causation of the disease. For instance, we have been taught to regard the existence of atrophy, commencing in the small hand-muscles, accompanied by fibrillar tremors, and late reaction of degeneration in affected muscles, as pointing certainly to some lesion of the cells in the grey matter of the anterior cornua. On the other hand, in an undoubted myopathy, such as pseudo-hypertrophic muscular paralysis, so called, we have never expected to find either fibrillar tremor or reaction of degeneration; and it is on those signs that diagnosis has hitherto been largely based. As will be seen, when studying Schultze's latest observation, we can no longer, apparently, rely with certainty upon these signs, and unfortunately we have not yet any to take their place.

There have always been authors who have maintained that all cases of idiopathic muscular atrophy are due to primary disease of the muscles, the nervous changes being, according to them, secondary. Such is the opinion of Friedreich, Schultze, Lichtheim, Liebermeister, and others. A fatal objection to the universal adoption of this theory is the fact, that we may have the most advanced atrophy of the muscles, with an absolutely normal condition of the nervous system.

It would appear to the impartial student that there may exist, under the comprehensive head of progressive muscular atrophy, a number of forms having the common characteristic of atrophy, but due to various causes. Thus we may have atrophies of *myelopathic* origin, due to disease of the anterior horns of the

spinal grey matter; or we may have *myopathic* atrophies, in which the seat of disease lies *solely* in the muscles themselves; or lastly, we may have *neuropathic* atrophies, in which we must look for the lesion in the *nerves* themselves. The cases hitherto recorded may most of them be classed under these three heads, but it must be admitted that there are still many anomalous cases.

There has not been added much that is new concerning myelopathies; but the recognition of the two other groups above mentioned has had the effect of considerably limiting their number, and at the same time of making their symptomatology more sharply defined. Thus the *thenar form* has become the type of a myelopathic atrophy, and though apparently in some true myelopathies the atrophy may commence in other than the hand-muscles, the number of such atypical cases is becoming less and less. The commencement of the disease in the small hand-muscles, at first sight so difficult to understand, has had great light thrown upon it by the work of Ferrier and Yeo[1] on monkeys, by which it is shown that the small hand-muscles are connected with centres in the dorsal cord at about the level of the second dorsal roots. Beevor[2] has illustrated the clinical bearing of this in myelopathies. From these facts we may speak of the thenar type of progressive muscular atrophy as a poliomyelitis, attacking first the anterior grey cells in the region of the upper dorsal roots and spreading upwards and downwards from that focus. It is, however, highly probable that a simple protopathic lesion of the grey cells, uncomplicated by any affection of the white matter, is much rarer than is usually supposed. This is Schultze's[3] opinion, and he goes so far as to assert that only two such autopsies are as yet recorded, namely those of Charcot and Gombault,[4] and Pierret and Troisier.[5] It is a fact that in many recorded autopsies there is coincident affection of the white matter.

It is in the class of atrophies in which the seat of the disease lies wholly in the muscles themselves, that the most important advances have been made. One of the earliest recognised myopathies is pseudo-hypertrophic muscular paralysis. It is needless to dwell on the symptomatology of a disease so well known, but it will not be out of place to mention the grounds on which the diagnosis of a myopathy from a myelopathy has hitherto been based.

As a rule, *fibrillar tremors* are not present in myopathies, it is true that in Schultze's[6] case, to be mentioned more fully later on

[1] Ferrier and Yeo, 'Proc. Roy. Soc.,' 1881.

[2] Beevor, 'Med. Chr. Trans.,' 1885.

[3] Schultze, 'Ueber den mit Hypertrophie verbundenen Progressiven Muskelschwund,' etc.: Wiesbaden, 1886.

[4] Charcot et Gombault, referred to by Charcot in 'Maladies du Système nerveux,' t. ii., p. 206, 4e éd.

[5] Pierret et Troisier, "Note sur deux cas d'atroph. musc. progr."—'Arch. de Phys. norm. et path.,' 1875, p. 236.

[6] Op. cit.

they were noted at an early period of the disease; but this is very exceptional, and when these little tremors are present, we may, with tolerable certainty, exclude myopathy pure and simple.

Till quite lately, it was believed that reaction of degeneration in the atrophic muscles was a sign of some nervous lesion, central or peripheral, and that it was never to be found in simple affection of the muscles. This, however, can no longer be relied on, for Zimmerlin, Schultze, Landouzy and Déjérine have shown that it may be present in pure myopathies. *Hypertrophy* of certain muscles is very often present in myopathies, but has never been seen in myelopathies or neuropathies. Very important points in forming a diagnosis are, the age at onset, the point of attack, the existence or non-existence of heredity, which is very common in myopathies and neuropathies, but not in myelopathies.

A myopathy nearly allied to pseudo-hypertrophic paralysis is the so-called "Juvenile Form," first recognised by Erb in 1884.[1] This form seems to have obtained general acceptance as a distinct type. It differs from pseudo-hypertrophic paralysis in the fact that hypertrophy is a very subordinate feature, while atrophy is a prominent one; in fact, hypertrophy may be sometimes absent, or so slight as to escape observation. The disease begins in early adolescence, and there is a strong tendency to heredity. The muscles first affected are those of the shoulder-girdle. As a rule, degenerative electrical reactions are absent, but this is not invariable. Fibrillar tremors are conspicuous by their absence. Erb's description is based on the clinical observation of twenty cases. Lichtheim,[2] however, has recorded a case rather closely resembling Erb's, in which absolutely no disease of cord or nerves were found.

Schultze's[3] cases are very interesting. There is a marked heredity. Maternal grandfather and grandmother were cousins, but quite healthy; of their children, one son had muscular atrophy of some sort; a daughter, who was quite free from any nervous or muscular affection herself, and was married to a man equally healthy, had five sons, three of whom have muscular atrophy; one died, and an imperfect record is given of the other two. In both the disease began at about eight years. The elder, in whom the disease was less advanced than in the younger, presented the "clinical picture" of pseudo-hypertrophic paralysis, that is to say, the disease began in the calves, which were much hypertrophied. He had the characteristic gait, and difficulty in rising from the ground. But there was also atrophy of other muscles, particularly about the shoulders. The younger brother showed a much more wide-spread atrophy, but without any hypertrophy, and presented the appearance described by Erb.

[1] Erb, "Ueber die Juvenile Form der progressive Muskelatrophie."—'Deutsches Arch. für Klin. Med.' Bd. xxxiv., 1884.

[2] Lichtheim, "Progress. Muskelatroph. ohne Erkrankung der Vorderhörner des Rückenmarks."—'Arch. für Psychiatr.,' 1878, p. 521. Abstract in 'BRAIN,' Vol. II. p. 142.

[3] Schultze, "Hereditäre Muskelatrophie und Pseudohypertrophie der Muskeln."—'Neurolog. Centralblatt,' 1884, p. 529.

Zimmerlin[1] records two families affected with myo-atrophy, much resembling Erb's form, but in one case there was reaction of degeneration in several of the atrophied muscles, and in another there had been fibrillar tremors in the earliest stage of the disease.

Marina[2] gives two cases of what he considers the juvenile form, but without heredity or hypertrophy. In one of his cases there is a partial reaction of degeneration (KCC = ACC) in the deltoid. Marie and Guinon[3] endeavour to show the close connection between the various forms of myopathy. Their first case is one which resembles pseudo-hypertrophic paralysis, but has no marked atrophy of muscles, nor any hypertrophy; but there was progressive weakness of leg-muscles, with the characteristic gait. At the same time the face-muscles seem to have been affected. The second case is one of Erb's form, and the last two belong to the "infantile form," to be described later.

Erb[4] gives an account of a case which he claims to be of the "juvenile form," but in which the muscular affection made its appearance after a fall on the back.[5] He excised a piece of muscle from the deltoid, which was hypertrophied, and another piece from the biceps, which was much wasted. In the former he found the muscle-fibres to be nearly all increased in size, very few showing signs of atrophy. The nuclei were much increased in number round the fibres, and there was also an increase of connective tissue. The vessels were thickened and had more nuclei in their walls than normal. The broadened muscle fibres showed no signs of fatty or granular degeneration, but their striæ were rather faint. In the atrophied biceps what fibres there were left appeared to be hypertrophied, and there was the same increase of nuclei as in the deltoid.

A most important study of muscular wasting is that of Schultze,[6] to which we have already referred several times. The work is divided into two parts, the special and general. The special part relates to a case which was one reported by Friedreich in his monograph on Progressive Muscular Atrophy. The history extends over a period of twenty years. There was a general widespread atrophy of the muscles of the upper extremities, including those of the hands, but also hypertrophy of some of the leg-muscles. There was affection of the diaphragm, but no bulbar symptoms. When

[1] Zimmerlin, "Ueber hereditäre (familiäre) progress. Muskelatroph."—'Zeitschr. für Klin. Med.,' 1884, p. 15. Abstract in 'BRAIN,' Vol. VII. p. 285.

[2] Marina, "Uno studio sulle amiotrophie."—'Lo Sperimentale,' 1885.

[3] Marie et Guinon, "Contrib. à l'étude de quelques-unes des formes cliniques de la Myopathie progressive primitive."—'Rev. de Méd.' 1885, p. 794.

[4] Erb, "Muskelbefund bei der Juvenilen Form der Dystrophia Muscularis progressiva."—'Neurol. Centralbl.,' 1886, p. 287.

[5] It is difficult to understand the connection between traumatism and the consequent occurrence of myopathic atrophy and hypertrophy. That such a connection does exist is shown by Eulenburg's case, recorded in the 'Deutsche Med. Wochenschr.,' 1885, p. 177, in which marked hypertrophy of the lower extremities, among other symptoms, followed a fall on the back.

[6] Schultze, 'Ueber den mit Hypertrophie verbundenen Progressiven Muskelschwund und ähnliche Krankheitsformen.' Wiesbaden, 1886. Abstract in 'Neurol. Centralbl.,' 1887, p. 13.

seen by Friedreich, fibrillar tremors were noted in the upper limbs, but none have been seen since. Reaction of degeneration was found in some of the atrophied muscles, such as the left deltoid, thenar muscles, and some interossei. The physical signs here are more in favour of the diagnosis of a myelopathy than that of a myopathy, and yet the anatomical appearances show that the case belongs to the latter class. Schultze found the nervous system, central and peripheral, quite normal, even the small intra-muscular nerves. The small blood-vessels in the nerves showed thickening of the intima, and in the brachial plexus and median nerve they were quite obliterated. The author does not claim anything peculiar to the disease in the occluded vessels, which he regards as due to general endo-arteritis. In the muscles important changes were found. He begins his description with the microscopical appearance of a muscle which appeared quite healthy to the naked eye. The muscle fibres in transverse section appeared angular, some were hypertrophied, but most of them were natural in size. There was an increase of sarcolemma and muscle nuclei, but, as a rule, very little increase of interstitial connective tissue. There was some fatty infiltration in the interstices between the fibres. Here and there were what looked like giant cells filled with smaller cells. These were the remains of muscle fibres, in which the true muscle substance was invaded by the proliferated muscle and sarcolemma nuclei. In muscles showing the highest degree of atrophy, and in which the reaction of degeneration had been made out, large infiltration of fatty tissue was seen, with a number of broad connective tissue fibres, in the meshes of which lay groups of nuclei, indicating the position of destroyed nuclei fibres. In a longitudinal section these nuclei appeared as long rows parallel to one another, sometimes consisting of a chain of forty nuclei.

In some of the muscles, especially the deltoid, the muscle fibres were undergoing a process of vacuolisation. Some fibres presented a sieve-like appearance from the numbers of small vacuoles, others had one large vacuole in the centre.

Dreschfeld[1] gives the results of an autopsy on a case which he believes to be Erb's myopathy. The brain and spinal cord were perfectly normal, as also were the intra-muscular and large nerves. The muscles showed simple atrophy of the fibres, with some increase of muscle nuclei.

Otto Buss[2] relates the case of two children, brother and sister, æt. 16 and 13 years, respectively. Father and mother are cousins. The author remarks that the boy has a stupid expression, but does not refer it to any affection of the facial muscles. Weakness was noticed first in the legs, æt. 10, but the shoulder girdle-muscles were soon found to waste. Hypertrophy was noted in part of the deltoid and triceps. No reaction of degeneration, or fibrillar

[1] Dreschfeld, "On some of the rarer forms of Muscular Atrophies."—'Brain,' July 1886.

[2] Otto Buss, "Zur Lehre von der Dystrophia muscularis progressiva."—'Berlin Klin. Wochenschr.' 1887, No. 4.

tremors. The girl showed a similar condition, but in a less degree. Some muscle was excised from the boy's biceps, and widening of the muscle fibres, together with increased sarcolemma nuclei, was noted. There was also increase of interstitial connective tissue.

Edgren[1] describes two cases, males, in whom the disease appeared at the ages of 16 and 22. They seem to belong to this type, but there was no heredity in either case.

This appears to be the best place to mention the conclusion of Roth,[2] based on autopsies of myopathies. He says that in excised portions of atrophied muscles he has long noticed that the individual fibres seem to end in long connective tissue fibres. This suggested to him the idea, that muscle fibres might atrophy in their length, perhaps, more than in their breadth. This, he maintains, is borne out by the clinical appearance of the atrophied muscles, the belly of the muscle becoming shorter and shorter. In one muscle that he examined, the zygomatic, he found that half the muscle consisted of tendinous fibrous material, the other half being natural. In a long muscle like the biceps, after suitable hardening processes a fibre could be stripped from one end to the other of the muscle, and then it was found that the part of it remaining of proper muscle tissue was only about 10–20 mm. long, while the rest was of pure fibrous tissue. He believes this to be peculiar to atrophies of myopathic origin.

Singer[3] gives an account of two complete cases, one of which belongs to this form, and the other to that of the type to be next described, showing how closely the two forms are allied. The first is that of a woman aged 34, single, with no heredity of a neurosis. The first symptoms were weakness of the lower extremities, with pseudohypertrophy of calves, and characteristic gait. This was followed by wasting of muscles generally. The shoulder girdle muscles were much atrophied, so also the upper arm muscles, but not the small hand muscles. No fibrillar tremors or reaction of degeneration.

Post-mortem.—The atrophied muscles showed general lipomatosis. There was some diminution in size of a small part of the lumbar grey matter on the left side, together with corresponding decrease in the number of ganglion cells, especially in the lateral bone, where on the right side there were twenty-six, and on the left three. Other nerves quite normal. The autological appearances of the muscles consist of increase in the interstitial connective tissue, splitting up of the fibres in a longitudinal direction, with also a tendency to split into plates horizontally, as occurs in every degeneration. The nerves were absolutely normal.

In the second case, the face muscles were affected on the left side, otherwise the case resembles the last, particularly in the

[1] Edgren, "Om den primära progressiva myopatin, etc."—'Nord. Med. Arkiv,' Bd. xix. Nr. 5.

[2] Roth, "Contribution à l'anatomie pathologique de l'atrophie musculaire progressive."—'Comptes Rendus de la Soc. de Biologie,' Dec. 17, 1886.

[3] Singer, "Zur Kenntniss der primären Myopathieen."—'Zeitschr. für Heilkunde,' Bd. viii. p. 229.

matter of the histological appearance of a piece of muscle excised for the purpose. Both these cases obviously are closely allied to pseudo-hypertrophic paralysis.

We now come to consider another distinct type of myopathy. Duchenne was well acquainted with a peculiar form of progressive muscular atrophy, in which the face muscles were early affected, and which so constantly appeared in childhood, that he called it the "Infantile form" of progressive muscular atrophy. The mode of attack is so characteristic, that instances of the disease may be traced in many of the descriptions of the earlier writers.

Landouzy and Déjérine [1] contribute a very valuable addition to our knowledge of this form. The first-named observer was so fortunate as to have been able to watch two brothers for a period of ten years.[2] In another family affected with the same disease, the authors were able to make a post-mortem examination on one of the members, by which it was established that the disease is purely myopathic, the nervous system, central and peripheral, being absolutely normal. In this disease there rarely if ever occurs any hypertrophy of muscles, atrophy is the predominant feature. The group of muscles first affected are those of expression; then soon follows atrophy of the shoulders and upper arms, the small hand-muscles being affected very late in the course of the disease. The muscles of the lower extremities may be affected sooner or later. As in the type last considered, fibrillar tremors are almost never present, and degenerative electrical reactions are rare. As the facial affection is peculiar, it will be well to describe it here. The forehead becomes smooth and quite devoid of wrinkles, even during vigorous alterations of expression, such as laughing and crying. Owing to wasting of the orbiculares palpebrarum the eyes are never quite closed, even during sleep. The lips are everted from wasting of the orbicularis oris, and the levatores labii superioris, but the zygomatics and risorius seem to escape, so that in laughing the mouth extends laterally (*rire en travers*), the upper half of the face not sharing in the change of expression. The muscles of mastication and deglutition escape entirely. The whole appearance is termed the "facies myopathica" by the authors.

In Remak's[3] case, which appeared at nearly the same time as Landouzy's preliminary communication, the face muscles were affected after the shoulders and upper-arm muscles. He considers it to be one of the juvenile type of Erb.

Mossdoff [4] records a similar case to Remak's, but with asymmetry

[1] Landouzy et Déjérine, "Myopathie atrophique progressive, sans Neuropathie."—'Rev. de Méd.,' Feb. 1885. A preliminary paper by Landouzy appeared in 'Comptes Rendus de la Soc. de Biol.,' 1884.

[2] Landouzy, "Deux cas de l'atrophie musculaire progressive de l'enfance."—'Comptes Rendus de la Soc. de Biol.,' 1874.

[3] Remak, "Ueber die gelegentliche Betheiligung der Gesichts-Musculatur bei der Juvenilen Form der progr. Muskelatrophie."—'Neurol. Centralbl.,' 1884, p. 337.

[4] Mossdorf, "Ein zweiter Fall von Betheiligung der Gesichts-Musculatur bei der Juvenilen Muskelatrophie."—'Neurol. Centralbl.,' 1885, p. 1.

of the facial affection. Both these cases correspond to the descriptions of Landouzy and Déjérine.

Charcot's[1] case illustrates well the facial affection in this disease. He insists strongly on the close analogy between the three forms of myopathy, pseudo-hypertrophic paralysis, Erb's form, and the form now under consideration.

Westphal's[2] third case is one of facial myopathy, in which there is a marked atrophic heredity, younger sister, father, and uncle.

Cenas and Douillet[3] record three cases in the same family, two brothers and sister; the eldest appears to be a case of the type now under consideration. The next was the sister, in whom the atrophy commenced in the thenar muscles of the right hand, and that after suffering for six months or more from severe neuralgic pains in the arm. She had previously, some six years before being seen, had suppurative synovitis of the right index finger. This may have been the determining cause of the atrophy, but however that may be, the case reads like one of the Aran-Duchenne type. The last case, another brother, presented the facial appearances of a myopathy, but fibrillar tremors were seen in some of the muscles. The existence in the same family of myopathy and myelopathy is very interesting, and would point to a possible common origin for both. In this connection may be mentioned Philip's[4] observations. Here we have a family of eight children, four of whom were affected with pseudo-hypertrophic paralysis of the most marked description. On the mother's side there were a cousin and an uncle "paralysed." The father's family history was good, but he himself suffered from a form of spastic paralysis, with wasting and contracture of muscles, which seems to be undoubtedly of spinal origin, but which had probably connection with an accident.

In Ladame's[5] case, the grandfather on the father's side was said to have died of muscular atrophy. The patient was a young man in whom the disease commenced at the age of twelve, after typhoid. The muscles first affected were those of the shoulder and upper extremities, the face muscles being attacked later. Leg muscles were much wasted, knee-jerk abolished. There was no hypertrophy, or reaction of degeneration.

Westphal[6] records three cases, mother, daughter, and mother's

[1] Charcot, "Révision Nosographique des Atrophies Musculaires progressives."—'Progrès Médical,' 1885.

[2] Westphal, 'Berlin Klin. Wochenschr.,' 1885, p. 617.

[3] Cenas et Douillet, "Deux cas de myopathie atrophique, type Landouzy-Déjérine, et un cas d'atrophie musculaire, type Aran-Duchenne, dans la même famille."—'Loire médicale,' 1885, Nos. 7, 8.

[4] Philip, "Primary Spastic Paralysis, and Pseudo-hypertrophic Paralysis in different members of the same Family, with probable Heredity in both."—'Brain,' Vol. VIII. p. 520.

[5] Ladame, "Contribution à l'étude de la myopathie atrophique progressive."—'Rev. de Méd.,' 1886, p. 817.

[6] Westphal, "Ueber einige Fälle von progressive Muskelatrophie mit Betheiligung der Gesichtsmuskeln."—'Charité Annalen,' xi., 1886. Abstract in 'Neurol. Centralbl.,' Jan. 1887.

sister, evidently of this type of myopathy. The face muscles were markedly affected in all.

Landouzy and Déjérine[1] add six more cases to the number.

Obs. I. is a very characteristic case of the disease. The facial peculiarities with atrophy of the shoulder muscles existed in the mother, the maternal grandmother, also in the younger brother.

Obs. II., a young woman, æt. 27, had the "facies myopathica" well-marked, but there was no heredity, no fibrillar tremors, or hypertrophy; but there was reaction of degeneration in the orbicularis oris, and the flexors of the left forearm. The interest in Obs. III. lies in the fact that his brother was seen, described, and photographed by Duchenne in 1872, as an instance of the infantile form of progressive muscular atrophy. In Obs. IV. the face muscles were affected late in the disease, and degenerative reactions were found in several muscles. Obs. VI. was one of the scapulo-humeral type, but without the facial characteristics. The author made a post-mortem examination on this case, and found that the nervous system, central and peripheral, was perfectly normal. In the muscles they found appearances closely resembling those described by Schlutze (op. cit.). They consider these appearances to be due to an "irritative myositis."

Kreske[2] records a typical case of this disease, the facial symptoms dating from three years old. No hypertrophy, or reaction of degeneration.

From the foregoing cases and opinions of writers, it is easy to see that a very close analogy exists between the three myopathies, pseudo-hypertrophic paralysis, the "Juvenile form," and the "Infantile form." The first and third are clinically very unlike each other, but the second stands as a transition form between the two. For instance, hypertrophy is the predominant feature in the first, a very subordinate one in the second, and absent in the third. The second and third may so resemble one another as to be undistinguishable but for the facial symptoms in the latter. All three are diseases of youth, and all are very strongly open to hereditary influence. As far as they have been yet studied, the same essential morbid processes are at the seat of each. And yet with all these points of resemblance, they are still distinct nosological types. It is to be hoped that in the near future they may receive some less clumsy and misleading names than they do at present; the name "facio-scapulo-humeral type of myopathy," suggested for the last by the French authors, scarcely recommends itself either on the score of elegance or brevity.

Among the hereditary forms of progressive muscular atrophy, it has long been noticed that the disease may commence in the

[1] Landouzy et Déjérine, "Nouvelles recherches cliniques et anatomo-pathologiques sur la myopathie atrophique progressive."—'Rev. de. Méd.,' 1886, p. 977. Abstract in present No. of 'BRAIN.'

[2] Kreske, "Ueber die myopathische Form der progressive Muskelatrophie Betheiligung der Gesichtsmuskeln."—'Münchener Med. Wochenschr.,' No. 15, 1886. Abstract in 'Neurol. Centralbl.' 1886, p. 502.

lower extremities. This has been observed by Bamberger,[1] Friedreich and Leyden. It must be remembered that pseudo-hypertrophic paralysis generally commences in that way, but the form now to be considered has no relation to that disease at all. This form was recognised as a distinct type by Charcot and Marie[2] in February 1886, and almost at the same time, and quite independently, by the writer of this review.[3] The latter proposed for it the provisional name of the "peroneal type of progressive muscular atrophy," because though it may attack any group of the lower leg muscles first, yet it seems to commence oftener in the peroneal group than in any other.

In this form heredity is a marked feature; the disease generally makes its appearance in childhood. Though the leg muscles are first affected, Charcot and Marie notice that the small hand-muscles are nearly certain to be affected sooner or later. A very common early symptom is the appearance of talipes varus, due simply to loss of power in the peronei, not to contraction of opposing muscles; this talipes is very gradual and progressive in its development, unlike that due to infantile paralysis. The affected limbs are cold, and often tend to become livid from disturbance of vaso-motor influence. Cramps and pains in the limbs are of frequent occurrence. Fibrillar tremors may or may not be present. Reaction of degeneration, as a rule, makes its appearance sooner or later. In the thesis above mentioned, the writer has collected some twenty recorded cases from various sources, which appear to be illustrations of this type. Among these are accounts of four post-mortems by Virchow,[4] Oppenheimer,[5] and Friedreich,[6] which show that disease of the cord is absent in two, and in the other two what disease there is, is probably secondary, being situated in the posterior columns. On the other hand, in three of these autopsies was found a well-marked interstitial neuritis affecting the nerves supplying the atrophied muscles. It is therefore probable that this type is a true *neuropathy*, using the term neuropathy in its restricted sense.

Among the later recorded cases which appear to belong to this form are those of Osler, Schultze, Ormerod, and Charcot and Marie.

In Osler's[7] case heredity is remarkably shown, and can be traced backwards through three generations. The disease began unusually late in life, æt. 47, but the symptoms seem fairly

[1] Bamberger, "Bemerkungen über progr. Muskelatroph."—'Oesterr. Ztg. für prakt. Heilk.,' No. 7, 1860. Abstracts in Canstatt's Jahresb., 1860, p. 88.

[2] Charcot et Marie, "Sur une forme particulière d'atrophie musculaire progressive," etc.—'Rev. de Méd.,' 1886, p. 97.

[3] Tooth, "The peroneal type of progressive muscular atrophy." Graduation Thesis, M.D. Cambridge, 1886.

[4] Virchow, "Ein Fall von progr. Muskelatrophie."—'Virch. Arch.,' 1855.

[5] Oppenheimer, "Ueber progr. fettige Muskelentartung." Heidelberg, 1885. Abstract in Canstatt's Jahresb., 1855.

[6] Friedreich, "Ueber progr. Muskelatroph.," 1873. Cases I. and II.

[7] "On Heredity in Progress. Musc. Atroph., as illustrated in the Farr family of Vermont."—'Arch. of Med., N.Y.,' 1880.

characteristic. Schultze's[1] cases are those of two sisters and a brother, æt. 3, 5, and 9 years respectively. In them the disease commenced in the peronei, at 8 years, leading to very early and gradual appearance of talipes varus. In the eldest the thenar muscles were affected early.

There were no fibrillar tremors, no pains along nerves or in muscles, but complete reaction of degeneration in many of the muscles. Schultze himself considers the disease to be essentially due to a peripheral lesion. That it is not an ordinary neuritis would seem to be suggested by the collateral heredity.

Of Ormerod's[2] cases, two, a brother and sister, were affected with atrophy, and contraction of the peroneal and calf muscles, soon after measles; the hand muscles were attacked about four years later. There was reaction of degeneration in some of the muscles, but no fibrillar tremors. A very interesting fact concerning them is that the father has had atrophy of the right peroneal and calf muscles since three years old. He has been told that he had measles badly when he was a year old. The atrophic condition has not progressed in his case.

Charcot and Marie (op. cit.) contribute five cases, two of which are brothers. In all, the disease commenced in the leg muscles; peronei or calf, leading three cases to talipes. At a time varying from two to five years after, the hand-muscles were attacked. There was reaction of degeneration of some of the muscles in three of the cases, and fibrillar tremors in all. Cramps of the muscles were very common.

This form of muscular atrophy requires much more attention than it has yet received, and post-mortem examinations are particularly needed.

Seppili[3] tabulates amyotrophies as follows:

A. *Neuropathic.*—1. Peripheral (toxic, infectious). 2. Central (myelopathies). *a.* Progressive muscular atrophy. *b.* Amyotrophic lateral sclerosis. *c.* Deuteropathic spinal amyotrophies (from extension of disease into anterior horns).

B. *Myopathic.*—Progressive muscular dystrophy, or progressive primary myopathy. *a.* Infantile progressive muscular atrophy. *b.* Erb's juvenile form of muscular atrophy. *c.* Muscular pseudo-hypertrophy. *d.* Leyden's hereditary muscular atrophy. *e.* Transitional forms.

[1] Schultze, "Ueber eine eigenthümliche progress. atroph. Paral. bei mehren Kindern derselben Familie."—'Berlin. Klin. Wochenschr.,' No. 41, 1884.

[2] Ormerod, "Muscular Atrophy after Measles in three members of a Family."—'Brain,' Vol. VII.

[3] Riv. Sper. di Fren., p. 120, 1887.

BRAIN AND EYE—OPTIC NEURITIS.

BY JAMES ANDERSON, M.D.

It is now six years since the classical discussion on Optic Neuritis in Intracranial Disease took place during the first session of the Ophthalmological Society. Much work, clinical, microscopic and experimental, has since then been done in elucidation of the subject; but it cannot be regarded as much less of a *quæstio vexata* now than it was six years ago. Another more purely ophthalmological question has also been under discussion during these years, viz. Sympathetic Ophthalmitis. Notwithstanding important differences, the two questions have much in common, and it seems probable that when the one has been solved, the other will present but little difficulty. While in both cases the ultimate decision must rest on clinical observation, in both cases experimental observation can do much in clearing the ground, and in suggesting at least a *vera causa*. To Deutschmann, of Göttingen, we are indebted for experimental work on both subjects, and we purpose giving some account of his recent monograph on "Optic Neuritis in connection with Brain Disease,"[1] with some reference to the work of others, confirmatory and opposing.

Observers are now, and indeed they were six years ago, practically unanimous in regarding optic neuritis as a single process, not distinguishable into "choked disc" and "descending neuritis," as was formerly made out. It is clear on reading various descriptions of what were considered the distinguishing features of "choked disc," that there was no general agreement on the point. The term implied a theory, but expressed no clinical fact, and it has simplified matters that it is now practically obsolete. At the same time it would be very far from right to conclude that there are not types of neuritis. The neuritis of tubercular meningitis, for example, is as a rule of quite a different type from that of cerebellar tumour. The low, pinkish-white swelling of the first is very

[1] Jena, Gustav Fischer, 1887, p. 68. For full Abstract v. 'Ophthal. Review,' April, 1887.

distinct from the high, blood-coloured and generally blood-stained swelling of the other. Both however, are almost certainly but different intensities of a similar process, and probably the main element in the difference is the "choking," which, as shown by Gowers in the above-mentioned discussion, does actually take place, not at, but in front of, the lamina cribrosa.

In his paper, opening the discussion on Optic Neuritis, Dr. Hughlings-Jackson referred to three hypotheses regarding the cause of the inflammation: Von Graefe's, Schmidt's, and the reflex vaso-motor theory of Benedict, Brown-Séquard and others. The last hypothesis he considered, and the writer believes he still considers, the most plausible explanation, although he in no way commits himself to it. There are many difficulties in the way of accepting it. Reflex vaso-motor irritation, will, we presume, be readily admitted by all pathologists; but very few, we imagine, would admit that this irritation can proceed to actual inflammation, exudation of lymph, and hæmorrhage, such as occurs in tumour neuritis. The only instance at all parallel with it is that of Sympathetic Ophthalmitis, and the work of Leber, Deutschmann and others has made it highly probable that there is in every such case an infective process by way of the chiasma. The fact mentioned by Dr. Jackson, that tumours may induce discharges in neighbouring grey matter, goes only to show that tumours may cause an instability of grey matter, either by immediate pressure, or by interference with vascular supply, whether directly or reflexly. Von Graefe's theory attributes the neuritis to compression of the cavernous sinus by the tumour, the consequences of this compression appearing specially at the lamina cribrosa from its rigidity. The theory is negatived by the fact, that the ophthalmic and the angular veins communicate so freely that even obliteration of the cavernous sinus will cause no venous stasis in the eyeball or orbit.

Schmidt's theory attributes the neuritis to the pressure of fluid effused in the subdural and subarachnoid spaces, and forced into the vaginal spaces of the optic nerve; and Schultén has recently shown, that the injection of fluids into the subdural and subarachnoid spaces will actually cause narrowing of the retinal arteries and distension of the retinal veins, the changes however, being temporary. Deutschmann has subjected this theory to further experimentation in order to settle the question, what degree of hydrops of the optic nerve in animals is required to produce changes in the disc similar to those of the choked disc in man. With antiseptic precautions he laid bare the optic nerve, divided it, injected the distal part of it with agar-agar solution, and ligatured it,

taking care not to injure the central vessels. With moderate distension, greater however than that produced by cerebral tumour, there was only the temporary vascular change observed by Schultén, while in order to produce swelling and œdema of the papilla, the nerve sheath required to be so forcibly distended as to arrest the circulation in the retina, and even then the nerve showed no true neuritis. Such distension and arrest of circulation is never present in the human subject. Again, repeated injections of agar-agar solution into the cranial cavity of an animal, although they caused considerable intracranial pressure and distension of the optic sheath, in no instance produced inflammation of the papilla, the nerve or its sheaths. These experiments seem certainly to decide against Schmidt's theory.

In 1881 Leber put forward the view expressed in the Transactions of the Medical Congress of that year:[1] "The optic nerve is the path of communication between the affection of the brain and that of the eye. An essential part in this transmission is taken by the effusion of a serous fluid into the sheath of the nerve extruded from the cranium by the increased intracranial pressure. This fluid does not act by simple mechanical pressure, since its quantity is sometimes small; probably it possesses phlogogenic properties." To test this theory, Deutschmann repeated his injection experiments, using an infecting material in place of the agar-agar solution. A solution of common salt containing traces of staphylococcus was injected, in the manner above described, within the optic sheath. During the first day the papilla became red and swollen, the veins distended, the arteries perhaps diminished in size. Next day the papilla was greatly swollen, and the retina turbid; and the microscope showed well-marked neuritis and peri-neuritis. Injections of the same solutions into the skull caused acute inflammatory changes, resulting in death without the eyes being affected, and it was necessary, therefore, to select an infective material of slower action. A few drops of tubercular pus were introduced, with antiseptic precautions, into the subdural space. The animal remained apparently healthy for three weeks, when the papilla became red, and the veins tortuous. True papillitis followed, which either gradually subsided, or went on to post-neuritic atrophy. The microscope showed tubercular meningitis, the intracranial portion of the nerve normal, the orbital portion more or less altered, the sheath being distended more or less with exudation in the early stages, and occupied with tubercle deposits in the later; the nerve and its sheath being both inflamed. Deutschmann considers that this proves

[1] 'Abstracts,' p. 474.

Leber's theory as to the cause of neuritis, and he suggests that the phlogogenic elements contained in the fluid secreted in the subdural and subarachnoid spaces are micro-organisms already present in the system, but congregated round the tumour as a place of least resistance. Deutschmann considers that a true descending neuritis is rarely demonstrable, and when a continuous neuritis can be traced, he considers that it is generally an ascending, rather than a descending, neuritis; that the bulbar end of the nerve is affected first, and from this point it travels backward. Where only one eye is affected in intracranial tumour, Deutschmann suggests that there is in the one nerve-sheath some unusual obstruction, either recent, or of older date, preventing the passage of the infective material.

It will at once be seen that the evidence afforded by the injections of tubercular matter into the subdural space is not absolutely cogent, and Edmunds and Lawford point out that,[1] in order to substantiate his point, Deutschmann would require to show that if the animals be killed in the first few hours or days after inoculation, no optic neuritis is present; that at a later stage inflammation confined to the bulbar end of the nerve is found, and that later still the whole length of the nerve is involved. Edmunds and Lawford[1] have recorded the results of their examination of the optic nerves in cases of intracranial disease, and an analysis of cases of intracranial tumour with respect to the existence of optic neuritis. Having examined some forty cases, they found that in every case in which papillitis was seen during life there were inflammatory changes in the optic nerves in their entirety. In a few instances the inflammation was confined to the proximal part of the nerve, and in these the papilla, both to the ophthalmoscope and microscope, showed no changes, death having occurred before the inflammation had time to travel down the nerves as far as the papillæ. They conclude therefore that the optic neuritis which occurs in intracranial disease is due to the presence of a secondary meningitis, and they find themselves strengthened in this opinion by the result of their analysis of cases. Of ninety-six cases of fatal cerebral tumour, optic neuritis was present in eighty-six per cent. of the cases in which the new growth was situated in the basal ganglia or in the cerebellum, whereas in only forty-six per cent. of those cases where the tumour was at the convexity of the brain did neuritis occur. In the discussion on optic neuritis, Gowers stated that it was probable that mere distension of the sheath

[1] 'Ophth. Rev.' May, 1887.
[1] 'Ophth. Soc. Trans.' vol. iii. p. 138, and vol. v. p. 172.

might cause papillitis, but he showed by specimens that descending inflammation was almost invariably present, even where there was no naked eye evidence of it. One of his specimens is interesting in relation to Leber's theory. It showed, as usual, inflammation along the length of the optic nerve, but much more intense at the chiasma and behind the globe. From it he argued that a very slight degree of descending inflammation may be the connection between a neuritis of the cerebral, and one of the ocular extremity of the nerve. The optic papilla, as he remarked, is a structure in which inflammation readily occurs, whether from intracranial or from general disease. And it is to be remembered that the papilla may be repeatedly and severely inflamed. The writer has recorded a case of relapsing neuritis[1] where a patient recovered with perfect vision from two separate and severe attacks of neuritis. Since the case was recorded the patient has had a third attack similar to the others, and has again recovered with perfect vision. Such a case is somewhat difficult of explanation on Leber's theory alone; but Leber's theory does not actually contradict the theory of a descending neuritis, or peri-neuritis, as advocated by Edmunds and Lawford. It is possible, perhaps it is probable, that both views are correct, each cause playing a part, more or less, in different cases.

[1] 'Ophth. Rev.' May, 1886.

Reviews and Notices of Books.

The Functions of the Brain. By DAVID FERRIER, M.D., LL.D., F.R.S. Second edition. Rewritten and enlarged. London: Smith, Elder, and Co., 1886.

IT has been understood for some time by those who take even a general interest in neurological studies, that Dr. Ferrier has been engaged in preparing a second edition of his celebrated treatise on the 'Functions of the Brain'; and now that the work has made its appearance, it will receive a hearty welcome both from the profession and the educated public. In its external appearance, the new book is an almost exact reproduction of its predecessor, the green-coloured back of which formed such a prominent and well-known feature in the library of every educated physician. The new edition extends to 498 as against 323 pages in the old. If we consider that the author gives, not only a detailed account of the numerous researches he has himself undertaken in the ten years elapsed since the publication of the first edition, but also a systematic exposition of the results obtained by other investigators in the field of neurology during this period, it will be at once seen that he has exercised a marvellous amount of self-restraint in not increasing the size of his book by more than 175 pages; and when we come to examine the contents of the work, this self-restraint becomes strikingly apparent. The first chapter deals with the structure of the cerebro-spinal centres, and there is not a page of the fifty which it comprises in which the instructed reader will not observe the temptation under which the author must have laboured to branch out into discussions about contested points. Such discussions are useful and necessary under certain circumstances, but Dr. Ferrier judged, as we think wisely, that his book was not the proper place for them. He accordingly proceeds directly to his object, and gives a succinct and transparently clear account of the structure of the nervous system—an account which is as accurate and trustworthy as can possibly be imagined within the limits he has imposed upon himself.

Before proceeding further, we may notice the beauty of the original woodcuts which illustrate this chapter. The new edition is illustrated by 137 figures as against 68 in the old. All the illustrations are good; but the representation of sections of a lobule of the cerebellum by Dr. Beevor (Fig. 21), and of the medulla oblongata, pons, and corpora quadrigemina by Dr. Bevan Lewis (Figs. 14, 15, 16 and 19), are models of anatomical drawings, while the representations of sections of the cerebral cortex by Dr. Bevan Lewis (Figs. 28 to 33) are altogether unique,

and probably excel, in delicacy of drawing and beauty of execution, anything we have ever seen produced by the wood-cutter's art for the purposes of histological illustration. It is only those who have had the privilege of examining sections of the cortex cerebri, as prepared by Dr. Lewis or by one of his pupils, who can at all appreciate how wonderfully accurate and faithful these drawings represent the original sections. We cannot help expressing some regret, that these beautiful figures are not accompanied by a brief description of the histology of the cortex, even at the risk of adding a few more pages to the size of the book; but the omission is only another striking example of the self-restraint already spoken of, as being exercised by the author throughout every part of the work.

In the second chapter the author treats of the functions of the spinal cord. An account is given first of the course of the conducting paths of the cord, as determined by embryological and pathological observations as well as by experiments on animals; and here the author favours the opinion that the sensory paths lie, not in the posterior, but in the lateral columns, in the limiting zone of Flechsig. He admits, however, the difficulty this opinion encounters in the fact, that these areas do not undergo an ascending degeneration when the cord is divided transversely, and endeavours to surmount it by supposing that these tracts maintain a continuous connection with the cells of the posterior horns. But in that case the sensory fibres belonging to the lower extremities must be connected with the cells of the posterior horns throughout the whole length of the cord, and it is not clear how they can at the same time be situated in the limiting zone of Flechsig. The author believes that the muscular sense is not a simple sensation, but one formed by a combination of the common forms of cutaneous and muscular sensibility. He asserts that "the sensations arising in connection with muscular action are of a purely centripetal character," and utterly rejects Brown-Séquard's hypothesis, that the paths of the muscular sense pass in the anterior roots and motor tracts of the spinal cord.

The action of the spinal cord as an independent centre is next discussed; the functions of reflex activity, co-ordination of synergic movements, muscular and vascular tonus, trophic regulation, and the regulation of the movements of special organs like the bladder, rectum, sexual organs and iris, being passed in review. The remarkable sobriety of Dr. Ferrier's judgment in interpreting the phenomena resulting from destructive and irritative lesions of various parts of the nervous system, is apparent in his remarks on the experiments of Tscheschichin, Burdon-Sanderson, Wood, Ott, Aronsohn and Sachs, Eulenberg and others on the alteration of the temperature of the body which is caused by injuries of the spinal cord, medulla, pons, and certain parts of the brain. "The facts," he says, "only allow us to conclude that injuries of certain centres and tracts cause increased heat production, but the mode of operation is by no means satisfactorily indicated;" and "it by no means

follows that we are to assume the existence of a special heat-producing centre, distinct from those which influence the motor and vaso-motor apparatus in general."

In the next chapter, which deals with the functions of the medulla oblongata, the author throws out a very important suggestion with the view of explaining the devious course pursued by the vaso-motor nerves, and the dilator nerves of the iris, which, arising from their centres of origin in the medulla, descend through the whole length of the cervical part of the spinal cord to emerge with the upper dorsal nerves, whence they pass into the cervical sympathetic. He believes that the medulla ought to be regarded as the direct continuation of the dorsal spinal cord, and that the cervical portion is intercalated in the course of development for the innervation of the upper extremities. Many well-known embryological facts support this hypothesis, and if its truth be granted, an adequate explanation would be found for facts which are otherwise inexplicable; such as the course of the cardiac excito-motor branches of the sympathetic, the extraordinary path pursued by the spinal portion of the spinal accessory nerve, and the origin of the ascending root of the fifth nerve being as low down as the level of the fifth cervical pair of nerves.

The author next treats of the formations of the mesencephalon and cerebellum in a general manner, and classifies the functional manifestations of these organs under the heads of the maintenance of equilibrium, the co-ordination of locomotion and emotional expression. His discussion of the influence of tactile, visual, and labyrinthine impressions on the maintenance of the equilibrium and of the disturbances of equilibration caused by lesions of those impressions is exceedingly able, and demands a careful study. Dr. Ferrier now attempts to determine the functions of the different centres of which the mesencephalon and cerebellum are composed, but this task is so inherently difficult and complicated that it need not be wondered if it is only attended by partial success.

From the results of both irritative and destructive lesions of the corpora quadrigemina, he concludes that these ganglia "form an essential portion of the mechanism of the co-ordination of retinal and general sensory impressions with the mesencephalic motor apparatus concerned in the responsive adjustments of equilibration and the other adaptive reactions of which animals are capable after removal of the higher encephalic centres."

In considering the functions of the cerebellum, Dr. Ferrier passes in review the classical experiments of Flourens, and gives due weight to the staggering gait and other disorders of equilibration occasioned by destructive lesions of the organ, whether resulting from experimental injury or disease; but the most remarkable light on the functions of the organ is probably to be found in the definite movements of the eyes, head, and limbs which the author obtained from electrical stimulation of the cortex. It is, indeed, hardly possible to over-estimate the theoretical and practical value of Dr. Ferrier's experiments on the cerebellum, and it seems to us strange that they appear never to have been repeated by any

other physiologist, either for the purpose of testing the accuracy of the results already obtained, or of eliciting new facts by extending the scope of the experiments themselves. The author justly observes, that the ocular and other movements resulting from electrical excitation of the cortex of the cerebellum confer great significance on the phenomena observed in man, when a galvanic current is passed transversely through the skull in the cerebellar region. When the positive pole is placed in the right mastoid fossa and the negative in the left, the head and body sink towards the positive pole when the circuit is closed, and external objects seem to whirl to the left, or in other words, there is a conjugate deviation of the eyes to the right. The author seems, however, to have overlooked the similarity of these phenomena to those produced by pressure on the tragus of the ear, in two cases of Menières' disease reported by Dr. Hughlings-Jackson. In a case of the kind under the writer's observation at present, the patient, a young man aged 17 years, on putting the tip of the index finger of his right hand into the external meatus of his right ear, staggers to the left and backwards; there is at the same time a strong conjugate deviation of his eyes to the left, followed by a rapid oscillation of the eyeballs, the movements being accompanied by a subjective feeling of objects whirling to the right. The similarity of these phenomena to those resulting from passing a galvanic current through the head is too obvious to require pointing out. It seems probable therefore that in the galvanic experiment the current acts, not directly on the cortex of the cerebellum itself, but on the semicircular canals, and it is also probable that the negative pole is, as usual, the stimulating one, inasmuch as in our patient the inclination of the head and the drawing of the eyes were away from the ear which was the subject of irritation. But whatever view may be taken of the manner in which transverse galvanisation of the skull produces these ocular movements, it does not in the slightest degree detract from the great value of the results obtained by Dr. Ferrier from electrical excitation of the cerebellar cortex.

The author now comes to the consideration of the functions of the cerebrum, which forms by far the most important part of the work. Nevertheless, we do not propose to discuss the author's views on this subject at any great length. Our reasons are that his views, which were the subject of warm controversy ten years ago, are now almost universally accepted. Differences of opinion there may be with regard to minor details, but the great principles of cortical localisation are adopted by all neurologists, and daily acted on practically at the bedside. After describing the methods of investigation, Dr. Ferrier gives a detailed account of the phenomena resulting from electrical excitation of the cerebral hemispheres. The most important addition in the new edition as compared with the old, in this part of his subject, is to be found in the description of the movements observed by Horsley and Schäfer on excitation of the marginal convolution. These movements are, speaking broadly, caused by contractions of the muscles of the trunk, such as the thoracic, lumbar, and abdominal muscles, and of

those muscles of the limbs and head which have their origin in the bones of the trunk, such as the muscles which produce movements at the hip and shoulder joints and of the head. The movements in question are those which we have in common with the largest number of animals, and they are consequently the most general movements of the body, while the movements which are peculiar to man, such as those of articulation and of the hand in writing and in playing the piano, are organised in or near the operculum. Such a distribution of the localisation of the general and special movements of the body is not accidental, and an ample explanation of it will doubtless be found in the comparative anatomy and embryology of the brain, but into this wide subject we cannot at present enter. Other noteworthy facts which come out in this part of the enquiry are, that the fibres of the conducting paths which connect the motor area of the cortex and the spinal nuclei are excitable, and functionally differentiated like the cortical centres, whether they be examined in their passage through the centrum semi-ovale or in the internal capsule. The author has also found, that excitation of the corpus striatum causes tonic spasm of the opposite side of the body; while excitation of the optic thalamus is not followed by any motor manifestation.

Coming now to the interpretation of the motor reactions obtained on electrical stimulation, Dr. Ferrier believes that some of these movements indicate that the part of the grey matter irritated is directly connected with the motor strands of the crus cerebri and spinal cord, while others of the movements only indicate that the part stimulated is indirectly connected with these strands, the direct effect of the stimulation being some modification of consciousness. In accordance with this view, the author divides the excitable portion of the cortex into a motor and a sensory area, but which part is to be regarded as belonging to the one and which to the other, can only be decided by destructive lesions of the respective centres. If the centre is a motor one, its destruction will give rise to a paralysis of a definite group of muscles, and if sensory to some defect of common or special sensation.

Whatever theoretical objections may be urged against this division of the functions of the excitable area of the cortex, there can be no doubt that, regarded as a practical distinction, it has proved of immense value in Dr. Ferrier's hands, and one can only marvel how wonderfully near the truth his first surmises with regard to the localisation of the sensory centres have turned out to be. The visual centre, the localisation of which has been a subject of keen controversy, is now stated by Dr. Ferrier to be situated in the occipito-angular region, and not in the angular gyrus, as he first supposed. The author found that destruction of one angular gyrus caused temporary amblyopia of the opposite eye, while destruction of both gyri caused complete blindness, which partially disappeared in a few days. Vision was not appreciably affected by destruction of one or even of both occipital lobes. Destruction of the angular gyrus and occipital lobe in one hemisphere caused transient amblyopia in the opposite eye, and a more

or less enduring hemiopia in both eyes towards the side opposite the lesion, from paralysis of the retinæ on the same side as the lesion. Destruction of the occipito-angular regions in both hemispheres caused complete and permanent blindness. From these experiments, which are a model of cautious inductive enquiry, the author concludes that each angular gyrus is associated with both eyes, but chiefly with the opposite one, and that, through the occipito-angular region, each hemisphere is associated with the corresponding halves of both retinæ, thus proving that the semidecussation of the optic fibres which takes place in the chiasma is maintained in the cortical centres. Such being the author's conclusions, it is manifest that he is compelled to reject Charcot's scheme of the decussation of the optic tracts, as indeed we believe the distinguished author of that scheme has himself already done, with that freedom from bias and ready appreciation of new facts which characterises him. The scheme which Dr. Ferrier proposes to substitute for that of Charcot, and the less known ones of Grasset and of Sharkey, has the demerit of being somewhat complicated, but it is those who are unaccustomed to investigate nature that are most ready to talk about "the simplicity of nature's ways." Nature's ways, especially in cerebral physiology, are exceedingly complicated, and it is possible that there is no way of graphically representing the facts by a simple diagram; and the one proposed by Dr. Ferrier possesses the merit of representing the facts ascertained, both from experiments on animals and from pathological observation, with tolerable accuracy, and far better than any scheme which has hitherto been constructed. At the same time, we do not believe, and Dr. Ferrier would be the last to maintain, that we have yet had the final word on the localisation of the visual centre. It is probable that the recent observations of Spencer on the pineal eye of the Lacertilia is destined to throw a new light upon the structure of the visual central nervous mechanism, and each addition to the accuracy of our knowledge of the structure of nerve mechanisms will render more definite our knowledge of their functions.

In his previous edition Dr. Ferrier placed the centre for tactile and common sensation in the hippocampal region, but the recent observations of Horsley and Schäfer have proved that the gyrus fornicatus forms part of this centre. It has already been pointed out by Broca that the gyrus fornicatus and the gyrus hippocampus form one lobe, which he named the *falciform lobe*, and which he erroneously believed to be related to the sense of smell, but it now appears that this lobe forms the centre for tactile and common sensation. Whether it is or is not likewise the centre for the muscular sense remains to be determined. The localisation of the auditory centre in the superior temporo-sphenoidal convolution, proved by the early experiments of the author, and afterwards confirmed by pathological observations, is now universally acknowledged. Nothing is known with regard to the olfactory and gustatory centres beyond what was taught in the first edition of Dr. Ferrier's work, that both are situated in the hippocampal lobule and the lower temporo-sphenoidal convolutions.

Of the localisation of the motor centres we do not propose to speak. With the exception of the observations of Horsley and Schäfer with regard to the motor functions of the marginal convolution, nothing of great importance has been added to our knowledge of these centres beyond what Dr. Ferrier taught us ten years ago. We do, indeed, find that our knowledge of these centres is becoming more minute and definite as time advances. While formerly we heard of movements of the muscles of the shoulder, we are now taught that a destructive lesion of a very limited size in a particular part of the cortex causes paralysis of the biceps on the opposite side; and instead of finding that a particular area is related to the movements of the hand, we now hear of a thumb centre, and so on; but in principle and for practical purposes, recent investigations have not added much that is new to our knowledge of the localisation of these centres. This speaks well for the thoroughness with which the work was done by Dr. Ferrier in his first series of experiments.

With regard to the functions of the frontal lobe, Dr. Ferrier finds that electrical stimulation of the area comprising the base of the superior and middle frontal convolutions—the postfrontal area—gives rise to lateral movements of the head and eyes with dilatation of the pupil, while destruction of this area causes paralysis of these movements, resulting in a conjugate deviation of the eyeballs and rotation of the head towards the side of the lesion. The animal however soon regains the power of moving the eyeballs after destruction of the postfrontal regions, and even bilateral destruction of them causes only a temporary paralysis of the movements of the eyeballs and of the lateral movements of the head.

Electrical stimulation of the prefrontal area is not generally followed by definite motor manifestations, although Dr. Ferrier states that in two instances he observed movements of the eyeballs on irritation of these regions. Destructive lesions also seem to show that these regions are functionally related to the movements of the eyeballs and head. In one case of destruction of the postfrontal regions, in which the animal had completely recovered the power of moving the eyeballs and head, subsequent destruction of the prefrontal regions was followed by rapid oscillations of the head, apparent inability to turn the head, except *en masse* with the trunk, and drooping of the right eyelid. Although these symptoms also disappeared three days after the second operation, the author concludes that the "prefrontal regions belong to the same centres as the postfrontal, just as the occipital lobes belong to the visual centres." This conclusion is much strengthened by another line of enquiry. In one case, in which the prefrontal regions were extirpated, and the animal survived the operation for three months, a descending sclerosis was discovered in the innermost bundles of the internal capsule and foot of the crus cerebri on either side, but these degenerated tracts could not be traced beyond the upper part of the pons. In another case the postfrontal centres were first destroyed and seven weeks afterwards the prefrontal regions were removed. Two

months and a half after the second operation a descending degeneration was found in the innermost bundle of the internal capsule and crusta, the bundles most mesially placed showing a less degree of degeneration than those externally placed. This experiment shows that the innermost bundles are connected with the prefrontal region, and those which lie immediately external to them with the postfrontal centres.

These observations afford strong confirmation to the opinion already expressed by the author, that both the prefrontal and postfrontal areas are concerned with the movements of the head, and eyes. Dr. Ferrier finds from his recent as from his earlier experiments, that although extirpation of the prefrontal areas is not followed by any manifest sensory or motor disorder, yet the animals operated upon undergo a decided mental deterioration, and in this conclusion he is borne out by the results obtained by Horsley and Schäfer, and Hitzig and Goltz.

The experimental investigation of the functions of the basal ganglia is surrounded by innumerable difficulties, owing to their complexity, and to the serious injuries which must necessarily be inflicted on other important parts in gaining access to them. The fact that electrical excitation of the corpus striatum gives rise to a tonic spasm of the opposite side of the body, shows that it forms part of the motor nervous mechanism, but Dr. Ferrier rejects Meynert's opinion, that it is a "ganglion of interruption" of the cerebral motor conducting paths. He adopts the view, which is now more commonly received, that this ganglion is an independent centre of the same nature as the motor centres of the cerebral cortex; in his own words, "the corpora striata proper are centres of innervation of the same movements as are differentiated in the cortical motor centres, but of a lower grade of specialisation." Much less is known of the functions of the optic thalamus than even of those of the corpora striata. The facts that irritation of this ganglion is not followed by any motor manifestation, and that when destroyed its fibres undergo an ascending degeneration, warrant the conclusion, that it has some relation to sensory centres and tracts; and it is also probable that the posterior part of it has a special relation to the visual sphere. "It is probable," says Dr. Ferrier, "that the optic thalami, specially related to the sensory tracts, and the corpora striata, specially related to the motor tracts, represent in a subordinate manner all the sensory and motor centres of the cortex, and constitute together a sensorimotor mechanism, subservient to the manifestation of all those forms of activity which do not imply conscious discrimination or true volition."

The author now considers the functions of the hemispheres from a psychological standpoint, and ably discusses such vitally important questions as the connection between brain and mind, the conditions of perception, the feelings and emotions, the appetite and desires, the motives to volition, the growth of volition, the conflict of motives, the acquisition of speech with the related subject of the dissolution of speech or aphasia, the control of

ideation, and attention, with a consideration of the anatomical substratum of this mental act.

A mere enumeration of these subjects shows how impossible it is to enter in this place upon a profitable analysis of Dr. Ferrier's treatment of them. The reasoning is so close that the student of psychology cannot afford to lose sight of a single sentence; and even the busy practitioner will find the masterly manner in which the intricate subject of aphasia is here presented before him of the greatest practical value.

The work concludes with a chapter on cerebral and cranio-cerebral topography, which is improved as compared with the corresponding part of the first edition by the introduction of Reid's admirable diagrams of cranio-cerebral relations.

In bringing our remarks to a close we have no hesitation in saying, that this work is one of which it is scarcely possible to speak too highly. In its first edition it was not simply a treatise on the functions of the brain, but a record of a revolution, effected in great measure by the author's own labours, in one of the highest and most complicated departments of science. And if it is not given to the author to present to his profession and the public so much of a revelation in this as in the former edition, it is not because his right hand has lost any of its cunning, but because medical literature is now permeated with his principles and the medical mind has become saturated with his opinions. To master such a work as this is in itself an education, and whether it be studied with the view of observing what it is possible to achieve by skilfully devised and dexterously executed experiments, of noting the powerful dialectic with which fallacies are exposed and false conclusions demolished, or of following the author through subtle psychological distinctions and abstruse speculations, no one can rise from an attentive perusal of the work without feeling an accession of strength to his intellectual capacity, and being inspired with additional hope for the future of medical science. To recommend Dr. Ferrier's book to the notice of the readers of "Brain" would be a singularly superfluous task, and as for the author himself, his reputation is now so widely known and so firmly established that he can afford to feel alike indifferent to adverse or favourable criticism. It may at last be permitted to us to say that no one can pretend to undertake any accurate work in neurology, whether it be for the practical purposes of discriminating obscure diseases at the bedside, or of extending our knowledge of the functions of the nervous system by new researches, without having a copy of Dr. Ferrier's book by his side for frequent reference.

James Ross.

once thrown into a state of complete contracture. Prof. Charcot explains this fact by assuming an over-excitability of the motor spinal cells, and shows how similar effects may be observed after hemiplegia, and in certain cases of hysteria where percussion of tendons gives rise to persistent contraction of muscles.

Facial hystero-spasm.—Prof. Charcot describes a curious case of tonic spasm of the facial, with obvious psychical complications, in a hysterical girl. Treatment made things rather worse, until complete isolation was enforced.

IV. and V. *Atrophy and contracture in arthritis.*—Chronic articular rheumatism is divided by Charcot into: (1st) the primitive generalised or progressive type; (2nd) the primitive partial or fixed type; (3rd) Heberden's nodosities. The first usually begins in the metacarpo-phalangeal joints and thence spreads widely. The second remains localised in one or two large joints. The third (often mistaken as of gouty origin) affects chiefly the terminal digital articulations. The morbid-process in all is a dry arthritic nature; and the three types are connected by transitional forms.

A curious feature of the muscular atrophy from joint lesions is that it is often associated with exaggerated tendon-reactions. One even observes in some cases that percussion of the knee gives rise to muscular contractions in distant muscles. This muscular hyperexcitability is related to the contractures, the rigid flexed attitudes, often assumed by a limb affected with painful joint affection; and as Charcot pointed out more than thirty years ago, the singular deviations of bones observed in rheumatic arthritis are due to reflex muscular spasms. Thus both the amyotrophies and the contractures of joint affections depend upon a reflex central nervous disturbance, either of an inhibitory or of an excitatory nature.

Ophthalmic migraine.—In a general paralytic of the characteristic "congestive" type of Falret, each attack was preceded with the appearance of a scintillating scotoma, followed by hemiopia, pain, and vomiting. These symptoms form the "ophthalmic migraine" of French authors, which also includes as subsidiary elements paralytic, aphasic and epileptiform phenomena. At the outset, these fits of migraine were the only symptom of the coming mischief, and Charcot insists upon their grave premonitory character.

VI.–VIII.—These lectures illustrate and discuss several points connected with *hysteria in the male*—a subject which has recently attracted considerable attention in France. Klein, who gives statistics, says that it usually is hereditary and appears after puberty. Boys, however, may also be affected, as Prof. Charcot proves by describing several well-marked instances. Hysterical men are by no means necessarily effeminate; they often belong to the artisan or labouring classes. A case is here described in which a smith developed a characteristic hysterical contracture of the hand after a burn. The details of, and commentary on, the case, are well worth reading in the original. It is to be noted that hysteria in males is occasionally of the severe type, but more frequently is not characterised by violent convulsive or emo-

Leçons sur les Maladies du Système Nerveux, par J .M. CHARCOT: Tome III. 1 vol. 8vo. pp. 518. Paris, 1887.

THIS third volume of Lectures is in every way worthy of its predecessors. We recognise throughout the master's hand, who knows so well how to clothe the wealth of information he has to impart, in the lucid grace of his yet powerful diction.

The first lecture has a pre-eminently historical interest. It consists in an opening address delivered by Prof. Charcot on assuming the new chair of neurology created in the medical Faculty of Paris. For twelve years the founder of the great Salpêtrière School had, by word and deed, laboured to demonstrate the necessity of founding a department for the study of nervous diseases. It required all the influence of his strong will and eminent personality to overcome the multitude of obstacles—the influences of inert officialism on the one hand, of active jealousy on the other—placed in his way. At last the hour of triumph came; and everywhere, outside at least that agonosphere of petty personal interests that surrounds every great enterprise, men of science were unanimous in congratulating Prof. Charcot for his achievement, and the authorities for their wisdom.

We shall endeavour, in the following pages, chiefly to give a brief summary of the contents of this volume. Lecture II. deals with the subject of

Muscular atrophy after articular lesions.—The patient, whose case formed the subject of this address, presented considerable atrophy of the triceps extensor of the thigh, following a blow on the knee. The articulation itself never was the seat of any extensive alterations, though soon after the accident there had been marked loss of motor power. This case is typical of a large class in which arthritic disturbances are followed with atrophy of the extensors. This atrophy is simple, not degenerative, the affected muscles displaying no qualitative alterations in their response to both currents. When their atrophy is considerable they may cease to react; but even then a spark from the static machine often excites a contraction. Prof. Charcot explains the nutritive alteration in the muscle to a reflex paralysis of the motor spinal cells which form its trophic centre. He does not however explain why the arrest of the trophic spinal influence from an *organic*, should give rise to degenerative, from a *functional* cellular disturbance to simple, atrophy. This is a point worthy of further inquiries.

III. *Traumatic contraction.*—It is a well-known fact, that an injury in a subject predisposed to gout, rheumatism, and other constitutional diseases, may give rise to local outbreak of his constitutional malady. The same rule obtains in hysteria. One of the old stagers in the Salpêtrière, whose epileptiform attacks had for some years become less and less frequent, fell one day on a staircase and slightly injured her left ankle. The whole limb was at

unable to visualise, or depict on paper, even simple geometrical figures. He used to be able to learn by heart poetry with the greatest ease, fixing in his memory the image of the pages, and, when reciting, reading off as it were from the recalled sight of these pages. Now all this was changed, and he had to trust to the reviviscence of the words as heard or articulated, that is to say of auditory or motor impressions, in the process of recitation. Strange to say there were but very slight traces of word-blindness; he could read with much the same facility as before—a fact that constitutes a striking exception to the application of the "law of dissolution" to this case. There is a point in the mental condition of the patient which we think deserves to be commended to the attention of psychologists. Though the name of a person or of an object failed to call up the faintest trace of a visual representation, he clearly understood such a word. The explanation of this fact is, we think, to be found only in the theory, that what we call a name is a symbol standing for a complex state of consciousness, involving the associational activity of several or all the special sensory cortical regions, with more or less distinct innervation of the emotional sphere. Hence, even when the leading sense-representation is absent, the remainder of the total conscious process is sufficient for the carrying on of a train of thought. Do we not here have an experimental demonstration of the fact, that the use of abstract or general names is possible without any concurrent visual representation individualising the class which it represents?

The subject of Lecture XIV. is a nosographical revision of *Amyotrophies*, which will be found mentioned in Dr. Tooth's Digest in the present number of BRAIN.

XV. *Tremors* vary as to their rate, as well as to their occurrence, form, and amplitude. Thus in disseminated sclerosis and paralysis agitans they consist of between four and five oscillations a second. The rate of hysterical tremor is six or seven; whilst in alcoholism, mercurialism, general paralysis, and exophthalmic the vibrations are more rapid, eight to nine a second. Among *Choreiform movements* we must distinguish between the meaningless gesticulation of chorea proper, the motor phenomena of athetosis, posthemiplegic chorea, on the one hand, and the more systematized and rhythmical movements of the conditions known as saltatory, malleatory and rhythmical chorea, on the other. These symptoms are most frequently observed along with other hysterical manifestations.

Lectures XVI.–XVII. contain the instructive cases of three children, two brothers and one sister, in whom severe hysteria was determined by taking part in spiritualistic performances—a fact to be commended to the attention of certain dabblers in "psychical research," and more particularly to the author and readers of a recent volume: "Spirit-workers in the home circle; a narrative of psychic phenomena in family daily life."—Charcot takes this opportunity to insist again upon the paramount importance of complete isolation from home and friends in the treatment of hysteria. It is a remarkable fact that, whereas the little patients

tional outbursts. Somatic symptoms are often observed without any psychical accompaniments. As a parallel to the smith's case of this "hystérie fruste," Charcot describes that of a girl who likewise presented contracture of the hand after a slight injury, though she never had presented any "hysterical" tendency before that event.

IX. *Spinal disease after injury to sciatic nerve.*—A cabinet-maker, aged 40, received a blow just below the left buttock. From that time he suffered from symptoms of ordinary sciatica, with pressure points, and occasional fulgurating pains and muscular spasms. About three months afterwards the painful symptoms disappeared, but the paralysis and degenerative atrophy of the whole limb above and below the seat of lesion, became more pronounced. The bladder was involved, necessitating daily catheterisations. Incontinence of fæces, and impotence were also observed. Some of the muscles displayed the degenerative reactions; and there was also muscular atrophy in the right leg. Charcot explains these symptoms by assuming a myelitis set up by peripheral irritation.

X. A case of *double sciatica* in a cancerous subject is the theme of some considerations on the frequency and clinical symptoms of the invasion of the vertebræ by cancerous deposits; whilst another, of *cervical pachymeningitis*, is described with special reference to its successful treatment by the cautery, followed by surgical procedures calculated to restore the movements of the rigid lower extremities.

XI. and XII. These lectures contain a full account of, and a discussion on, a very perfect case of *word-blindness*. This form of aphasia in which the patient, though he may still be able to speak and write perfectly, is unable to realise the language value of written symbols, has long been recognised in Germany and England. But in France, where the influence of Broca's discovery of the "speech" centre, and the defective psychological doctrines of the School, stood in the way of further progress in the knowledge of aphasic symptoms, it is only quite recently that the powerful influence of Charcot has broken through the tradition and silenced prejudice. Even Magnan's clear teaching on this subject had scarcely secured a fair hearing. Now "nous avons changé tout cela," and the study of the various forms of aphasia is pursued with much energy and success by a number of French neurologists, supported on the psychological side by Prof. Ribot, and other writers of the same school.

XIII. In marked contrast with the last case stands one of *visual amnesia*, or loss of mental imagery, the description of which is full of interest to the psychologist as well as to the physician. The patient, a highly-gifted and educated man, belonging to the "visual type" of Galton (*i.e.* carrying out his mental processes chiefly by aid of visual representations) suddenly found himself deprived of the power of remembering or recognising objects *as seen*. The reflection of his own face in the glass became unfamiliar to him. Though a fair draughtsman before, he was

had convulsive attacks on meeting one another, or their father and mother, they at once improved when placed among the patients at the Salpêtrière, many of whom are the victims of the graver forms of hystero-epilepsy. There is evidently much exaggeration in the opinion often heard expressed in this country, that cases are "manufactured" by the contagious influence of example, in the Salpêtrière wards. Here we had three young children who developed the disease in the seclusion of their solitary home, and were cured when placed in the very centre of the so-called infection. It is to be remarked that Briquet and Charcot have, long before Weir Mitchell, found the curative value of isolation and moral control, apart from the often unnecessary adjuncts of massage and over-feeding. It is a pity that the latter should have taken such an undue importance in practice as to obscure the efficacy of the former, which are amply sufficient to cope with bad cases of hysteria, even where malnutrition exists. Medical men should at any rate remember that, in some cases, too energetic physical measures neutralise rather than enhance the good effects of the moral factors.

In Lectures XVII.–XIX. (and Appendix) the author returns to the subject of *hysteria in the male*, the relatively frequent occurrence of which is still doubted by many physicians. The reason of this scepticism is that they expect to find in males the mobility which is—erroneously—considered as a pathognomonic sign of the hysterical nature of certain symptoms in women. Hence persistent anæsthesia, a depressed psychical condition, have been considered incompatible with the functional nature of certain nerve troubles often observed in men of strong physique and hard working habits, especially after a shock or a blow. The careful study of the eight new cases now described will convince sceptics that their position requires reconsideration; and the evidence accumulated in the succeeding lectures will no doubt appear irresistible to every impartial reader.

Two cases of traumatic *hysterical brachial monoplegia* are exhaustively described and discussed (XX.–XIX.). The first relates to a cab-driver who a week after a fall on the right shoulder lost the use of his arm. The paralysis was complete; but the muscles, though flaccid, reacted normally to electricity. There was complete anæsthesia of the limb (excepting the fingers and palm), involving every kind of sensation, even that of position in space. The clinical characters and distribution of such an anæsthesia are incompatible with any known organic lesion of the brachial plexus, cord, or brain. Further, ophthalmoscopic examination revealed visual troubles, in the shape of contraction of the field and monocular polyopia. The hysterical nature of the latter was proved by the co-existence of macropia and micropia. A pencil placed close to the eye appeared single and very large; at 8–10 cm. it appeared double; at 15–20 cm. its image was two or three times smaller than normal. The face, as is usual in hysterical cases of this sort, was quite free, and the tendon reactions normal.

In the second case the patient, a bricklayer aged 18, lost the use

of the left arm three days after a fall on the shoulder, and presented a complete anæsthesia of the whole limb. The hysterical nature of the lesion was demonstrated by the coexistence on the left side of hemianalgesia, diminution of special sensation, contracted visual field, monocular polyopia, hysterogenous zones, and other signs. Prof. Charcot very forcibly brings into relief the parallelism between these paralyses, which might be described as caused "by traumatic suggestion," and those obtained in hypnotised subjects by verbal suggestion. The characteristics of both classes are identical, and moreover the distribution of the anæsthesia that accompanies them is the same. The limits of the latter differ from that observed after actual lesion of the plexus, inasmuch as they do not correspond to nerve provinces: they are bounded above and below by planes transverse to the axis of the limb.

The chief practical importance of the study of these cases is to be found in the light thrown upon the phenomena of "railway-spine" or rather of "railway brain," a subject ably discussed in Herbert Page's well-known book. The treatment adopted by Charcot deserves notice In addition to douches and electrisation, he first endeavours to raise the expectant attention of the patient by insisting on the curability of their paralysis. Then by means of active and passive movements, and dynamometric practice repeated several times daily, he endeavours to restore those mental representations of movements, or muscular sense ideas, upon the obliteration of which the inability to move the limb seems to depend. This plan is often crowned with remarkable success, the anæsthesia disappearing *pari passu* with the paralysis.

Further illustrations of functional nervous disorders in the male are given in Lectures XXIII. and XXIV., the occasion of which was a typical case of traumatic *hysterical coxalgia*, a condition well known in England since the masterly description of it given by Brodie. The patient was a sawyer, aged 55, the father of seven children, who never had presented any sign of nerve trouble, until he met with the accident (shock from a steam-engine) that determined the coxalgia. The characteristic system of Brodie's disease is the hyperæsthesia of the *skin* in the neighbourhood of the hip. This was well marked in the patient; in whom also movement or percussion of the bones determined a diffused pain radiating from the hip, followed by a kind of hysterical aura. Moreover the left side of the body was found to be anæsthetic to pricking and temperature, except at the elbow, hip and knee; with partial loss of muscular and special senses. Examination under chloroform confirmed the diagnosis as to the functional nature of this coxalgia of about eight months' standing.

It is a curious and important fact that in hypnotised subjects the suggestion of a fall on the hip produces symptoms exactly corresponding to hysterical coxalgia, including even the well-known referred pain in the knee-joint. We find therein a confirmation of the view, that the mechanism of shock in the production of certain nerve troubles is essentially similar, and depends upon the "traumatic suggestion" already mentioned. Moreover, light massage,

both in the case of traumatic and experimental coxalgia, was followed with temporary anæsthesia and complete flaccid paralysis of the limb.

In Lecture XXV. we find an instructive case of *hystero-traumatic contracture*. The patient, a strong man of 30, butcher by trade, received a blow on the left arm, which became anæsthetic and paralysed without the trace of rigidity. Two days after, he applied, on account of some local swelling, for relief at a surgical clinique, when the limb was put up in a plaster bandage. Six weeks after, on removal of the latter, a contracture had set in, that persisted for several months when the patient came to the Salpêtrière.

Volkmann has described a form of muscular rigidity produced by tight bandaging, and which he explains by assuming that a coagulation of the myosine takes place in consequence of the ischæmia. At any rate electrical examination in such cases reveals alterations in the muscles which also do not relax under the influence of chloroform. In the case shown by Charcot, opposite conditions were observed. That the contracture was of hysterical, and not merely spasmodic, nature was shown by the fact that it was extreme, did not relax at all during sleep, and was accompanied with superficial and deep anæsthesia. Moreover it is easy to produce similar contractures in cases of hysterical subjects with muscular hypertonicity, by applying to the limb a few turns of Esmarch's bandage, and so obtain experimentally the very condition observed in the present case. The patient eventually recovered the use of the arm; but the fingers remained flexed at an angle of 90° owing to the supervention of fibroid retraction, a complication not unknown in protracted cases of spasmodic contracture.

The last lecture (XXVI.) deals with *hysterical mutism in males*. One of the patients, whose case is described in the Appendix, has since occupied the attention of the medical profession in this country, where he went under the designation of "the Soho-Sleeper." Hysterical mutism usually follows on an emotion, a fit, or a sore throat; it persists from a few hours to days, weeks, months, or even years, and often disappears suddenly. It is characterised by complete aphonia. The patient cannot whisper, nor even imitate the articulatory movements of the tongue and lips. It is a pure and complete motor aphasia, and something more, as every kind of vocal sound is impossible. The other functions of language are intact. Like other hysterical symptoms it is usually accompanied with some characteristic signs of the neurosis (anæsthesia, &c.) and can be experimentally induced by suggestion, upon hypnotised hysterical subjects.

A. DE WATTEVILLE.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Outline of Contents of Papers which have appeared during the Quarter. By ALEX. HILL, M.D. Cambridge.

COMPARATIVE ANATOMY AND DEVELOPMENT.

1. Central Nervous System of Acephalæ. RAWITZ (*Jenaische Zeitschrift für Naturwissenschaft*, 1887: pp. 384–461, pl. xxv–xxix.)

After a short description of the macroscopic structure of the nervous system of certain mollusca there follows an account of the histological methods used, and then an exhaustive description of the microscopic characters of nerve-cells and nerve-fibres in these animals, illustrated with numerous figures.

2. The Anatomy of the Nervous System in the Gymnophiona. Dr. JULIUS WALDSCHMIDT (*Ibid.* pp. 461–476, pl. xxx–xxxi.)

This is an amplification in certain points of the description of the anatomy of this group of amphibia already published by Wiedersheim. With regard to the remarkable double olfactory nerves first pointed out by Wiedersheim, Walderschmidt gives it as his opinion that the ventral root is the homologue of the olfactory nerve of other vertebrates, and the dorsal root a later secondary formation, depending for its development upon the great relative importance of the sense of smell to these blind animals.

3. The Brain of the Wasp (*Vespa crabro et vulgaris*). H. VIALLANES (*Ann. des Sciences Nat. Zool.*, tome ii. pp. 1–100, pl. 1–6).

The whole of the prebuccal portion of the ganglionic chain is regarded as "brain." This, as in previous articles, the author divides into proto-cerebron, belonging to the first somite, and connected with the eyes; deutero-cerebron, belonging to the second somite, which bears the antennules, and presenting a characteristic

structure due to its "olfactory glomerule;" trito-cerebron, or brain of the antennary segment, which differs in no respects from the ganglia of the ventral chain.

The paper contains a detailed anatomical description of these parts, and also a historical summary.

4. The Development and Innervation of the Nerve-ridge (Nervenhügel) of Urodelan Larvæ. P. Mitrophanow (*Biol. Centralblatt*, May 1887, pp. 174–176).

5. Man's Structure as Evidence of his History. Wiedersheim (*Berichte der Naturforschenden Gesellschaft zu Freiburg i. B.* 1887; pp. 168–278.)

Includes an account of the characters which distinguish the Nervous System in man from that of other animals, and a discussion of the phylogenetic bearing of these features.

6. The Medullary Folds of the Chick. E. Béraneck (*Recueil Zoologique Suisse*, 1887, pp. 305—pl. xiv).

A description of folds and thickenings of the hind brain in the embryo chick, similar to those found in reptiles, and throwing light upon the segmentation of the vertebrate skull.

Corpus Callosum.

7. The Origin of the Corpus Callosum, a Contribution upon the Cerebral Commissures of the Vertebrata. Part II. H. F. Osborn (*Morph. Jahrbuch*, pp. 530–543, pl. xxv, 5 *figures in text*).

A continuation of Osborn's work upon the Cerebral Commissures, in which he has proved that they exist in all vertebrates, and are always arranged according to the same fundamental plan. The present paper treats of the Commissures as they are found in certain adult ophidia and aves, and also of their development in the frog and sheep.

8. The Microcephalous Brain of Hoffmann, in which the Corpus Callosum was absent. Onufrowicz (*Arch. für Psychiatrie*, pp. 305–328, pl. viii. and ix.)

Critical summary of all hitherto recorded cases. Anatomical description of this case, in which the Corpus Callosum was completely absent, illustrated with figures showing external appearance

and numerous transverse sections. Except for the absence of Corpus Callosum, Lyra and Olfactory Bulbs, the brain may be regarded as normal although small. The internal capsule was normally developed. Subject was an idiot 37 years old.

CORTEX.

9. The Smell-Centre. A Study in Comparative Anatomy. Prof. ZUCKERKANDL of Graz. (*Publ. F. Enke, Stuttgart*, pp. 124, 7 *lithographs and* 25 *figures.*)

The author gives a description of the typical formation of the cortex-margin in the region of the Cornu Ammonis, and in particular of the callosal convolution (Balkenwindung) first described as a separate structure by himself. He then traces the variations in this formation exhibited in a large number of animals. The comparative anatomy of the olfactory lobe is then traced, and it is shown that when it is reduced in size the lobus Hippocampi suffers a corresponding diminution. The arrangement of the mesial convolutions is then described; the limbic lobe and the inner and outer elements of the arcuate convolution (Randbogen) are brought into a scheme forming three concentric rings completed in front by the roots of the olfactory tract and the variations in the development of the several portions of these rings are described. From the small development of the Cornu Ammonis in anosmatic animals, and especially in the dolphin, it is inferred that this belongs to the smell centre.

10. Illustrations of normal and defective development of the Multipolar Cells of the Cerebral Cortex; of their degeneration in Senile Insanity, and of certain Albuminoid or Protoplasmic exudations commonly found in the neighbourhood of the junction of the white and grey matter of the Convolutions in cases of General Paralysis and ordinary Mania, in which the cases have been more or less acute. EDWARD PARKER, M.D. (*Journal of Mental Science, April*, 1887, pp. 20–25, *with illustrations*).

11. Macro- and Microscopic Investigations on the Nerve-centres. Dr. CASIMIRO MONDINO (*publ. Turin, Unione Tipografico-editrice*, pp. 70, *nine large coloured plates*, 1887).

Devoted to a consideration of the exact structure, connections and mutual relation of the claustrum, nucleus amygdaleus and cortex of the apex of the gyrus hippocampi.

12. A Note on the means of Topographical Diagnosis of Focal Disease affecting the so-called Motor Region of the Cerebral Cortex. V. Horsley (*Am. Journ. Mental Science*, 1887, pp. 342–369).

Localisation.

13. The Heat-centre in the Brain. Ott (*Journ. Nerv. and Mental Disease, N. Y.* 1887, pp. 150–162).

14. The Question of Cerebral Localisation, with consideration of Cerebral Disturbances of Vision. Reinhardt (*Arch. für Psychiatrie*, pp. 449–487).

Conclusion of paper which appeared in previous volume.

Cerebellum.

15. A Case of Atrophy of the Cerebellum, with Degeneration in the Medulla Oblongata and in the Spinal Cord (probably the result of Alcoholism). Dr. Schultze of Heidelberg (*Virchow's Archiv*, 1887, pp. 331–343).

The case occurred in the person of a man 39 years old. There was a general diminution in size of the cerebellum in all its diameters. Microscopical examination revealed an obvious loss in white fibres; a diminution in the number of cells of Purkinje; atrophy of the corpus dentatum. In the medulla both olives were degenerated. Throughout medulla and cord there were various alterations which are minutely described, but which were not in all respects such as would, according to most theories as to the connections of the cerebellum, be expected. Türck's column and the direct cerebellar tracts were intact. In the upper part of the cervical swelling there was a small patch of degeneration, on the right side, lying just in front of the crossed pyramidal tract.

General Physiology.

16. Remarks on Evolution and Dissolution of the Nervous System. J. Hughlings-Jackson, M.D., F.R.C.P., F.R.S. (*Journ. Mental Science*, April 1887, pp. 25–48).

Nerve-facial.

17. The Muscles innervated by the Facial Nerve in a young Gorilla. G. Ruge (*Morph. Jahrbuch*, 1887, pp. 459–530, pl. xxiv.)

This is a study of muscles, although illustrative of the distribution of the facial nerve.

Nerve Auditory.

18. The Question as to the Origin of the Nerve of Hearing, and the physiological meaning of the N. Vestibularis. Professor W. Bechterew (*Neurolog. Centralbl.*, 1887, pp. 193–198).

Presents a summary of the views held by various observers on this matter. The author maintains the opinions as to the connections of the auditory nerve which he published in 1885.

Spinal Cord.

19. The Localized (Punktförmig) Stimulation of the Frog's Spinal Cord. Sirotinin (*Arch. f. Anat. und Physiologie. Phys. Abth.* pp. 154–177, *with Plate II. giving illustrations of the Clamps used*).

The cord was stimulated by puncturing with a needle. The percentage number of times that movement of certain muscles (Ileo-psoas, Semitendinosus Gastrocnemius) resulted from the prick was observed. The region, puncture of which gave rise to the maximum number of movements of one of these muscles regarded as in especial connection with this muscle.

Latent period for contraction of each muscle, after puncture of each region from which it could be obtained, measured.

Cord surrounded by fourth vertebra associated with Ileo-psoas. Fifth vertebra semitendinosus. Sixth gastrocnemius. Comparison of these results with the effects of electrical stimulation.

Reflex Action.

20. A Crossed Reflex in the Frog. O. Langendorff (*Archiv für Anat. und Physiologie. Phys. Abth.* pp. 141–143).

Contains further details with regard to the manner of producing by means of an electric stimulus the crossed reflex already described by L. The reflex movement consists in a tetanic flexion and abduction of the opposite hind leg, when pressure is made with a blunt instrument behind the eye or over the tympanic region.

21. The One-sided and Both-sided Closure of the Eyelid. By the same Author (*Ibid.* pp. 144–147).

22. Contribution to the Study of Reflex Phenomena. The adapted and defensive Reflexes among Mammals from the results of a new experimental method. J. V. Laborde (*Comptes rendus de la Soc. de Biologie*, 1887, p. 65).

After removal of both hemispheres from new-born kittens or guinea-pigs by the water stream (Goltz's method), very extensive appropriate, and co-ordinated reflex actions can be obtained almost up to the time of death.

23. Reflex of the Spinal Cord in the Dog (*Ibid.* p. 110).

Spinal Ganglia.

24. The Physiology of the Spinal Ganglia. Dr. Max Joseph of Berlin (*Neurolog. Centralbl.*, 1887, pp. 172–175).

Experiments made on a number of kittens which were killed six to eight weeks after section of the second cervical nerve, either on the proximal or the distal side of the ganglion.

When the section was made between the cord and the ganglion, the results were just such as obtained by previous observers. When the nerve was cut beyond the ganglion, no change in this structure resulted, but a number of fibres degenerated both in their course through the ganglion and in the posterior root. The larger number of fibres of the posterior root remained intact.

Peripheral Nerves.

25. Termination of Nerves in the Liver. A. B. Macullum, B.A. (*Quart. J. Microsc. Science, March* 1887, pp. 439–460, pl. xxxii.)

Observations made with the chloride of gold method showed that there is an intimate relation between nerve fibrils and liver cells. Nerve filaments enter the cells (frequently dividing in a dendritic manner within their substance), and terminate in bead-like enlargements in the neighbourhood of the nucleus.

26. Some Variations in the arrangement of the Nerves of the Human Body. D. Hepburn (*Journ. Anat. and Physiology*, 1887, pp. 511–514).

27. Note on the Nerve-supply of the Musculus Sternalis. J. C. Lamont (*Ibid.* pp. 514–515).

Nerve Fibres.

28. Histology of crushed (Gequetschen) Nerves. Franz Tangl (*Arch. f. Mikrosc. Anat.*, pp. 464–470, pl. xxvii). (*Also in Hungarian in Oevosi Hetilap.* 1887, *Nos.* 2, 3, 5, 10 *and* 11.)

Behaviour of the axis cylinder after ligaturing of nerves shows

that it is not fluid, but has considerable powers of resistance and perhaps elasticity.

SYMPATHETIC.

29. Observations upon the persistent effects of Division of the Cervical Sympathetic. P. H. PYE SMITH, M.D., F.R.S. (*Journal of Physiology*, 1887, pp. 25–49).

A series of observations carried on systematically between 1878 and 1884.

CIRCULATION.

30. Physiological Diurnal and Nocturnal Variations in the Brain-pulse. RUMMO and FERRANNINI (*C. R. Acad. des Sciences, Paris* 1887, pp. 310–313).

STAINING.

31. Method of combining Weigert's Hæmatoxyline-copper stain with the use of the Freezing Microtome. D. J. HAMILTON (*J. Anat. and Physiology*, 1887, pp. 444–450).

Gärtner and Wagner on the Cerebral Circulation (*Wr. Med. Wochenschr.* Nos. 19 and 20, 1887).—The authors have gained important results by applying to the problems of the cerebral circulation a method of investigation which has been fruitfully employed in researches concerning the circulation in other organs.

The method consists in measuring the amount of blood which flows through an organ in the unit of time—which therefore passes away by the venous system. In this manner, it is possible to draw conclusions as to the innervation of the vessels of the organ concerned, due regard being allotted to certain other circumstances, especially the blood-pressure.

The authors' experiments were performed on dogs. The flow of blood was registered on a hymograph by means of a cannula placed in one of the external jugular veins, which, in dogs, convey the chief mass of the cerebral blood.

As the observations were limited only to one of the many cerebral venous channels, it was necessary to determine how far that limitation might vitiate the experiments. If an impediment to the outflow from the other exit channels were brought about by an increase in the venous pressure, more blood would flow through the prepared vein, and contrariwise with a sinking of the venous

pressure. But by making the resistance to the outflow in the prepared vein very small in proportion to that which obtained in the remaining effluent vessels, any error arising from the limitation referred to, and from fluctuations in the extra-cranial venous pressure, was reduced to a negligible quantity.

The arterial blood-pressure also had to be taken into account. It is clear that, the calibre of the brain-vessels remaining equal, so much the more blood must stream through them, the higher the pressure under which it circulates. Consequently a fluctuation in the outflow quantity can only be taken as evidence of a change in the calibre of the cerebral vessels, when that fluctuation is not adequately explained by a variation in the arterial blood-pressure.

The influence of the blood-pressure upon the cerebral circulation can be shown by simple experiment; *e.g.* the blood-pressure can be raised by compressing the aorta above the diaphragm. As soon as it be thus raised, the quantity of blood issuing from the cerebral vein increases in an exactly parallel manner to the blood-pressure. This increased outflow continues so long as the raised pressure is maintained, and ceases parallel to the blood-pressure when the compression of the aorta is discontinued. On the other hand, if the blood-pressure be considerably lowered by compression of the ascending vena cava, the outflow quantity immediately sinks; and when the blood-pressure falls under a certain limit, which lies at 30–40 mm. of mercury, the efflux of blood from the brain quite ceases. The same is observed when the pressure is lowered by bleeding.

To control this possible source of error, the arterial blood-pressure was registered throughout all the experiments, by aid of a cannula in one of the crurals.

Certain agents influence very energetically the blood-vessels of various organs through the vaso-motor centre. Such an effect can be brought about in a reflex manner or by toxic means, *e.g.* irritation of a sensory nerve, administration of strychnine. The vessels of the abdominal organs especially are contracted by such means, and there occurs in consequence a considerable raising of the blood-pressure. It was of interest to see whether the vessels of the brain also took part in this contraction.

If the cerebral vessels be contracted to the necessary degree, the quantity of blood passing through the brain must diminish, in spite of the increasing blood-pressure, as can be very strikingly shown under like circumstances in the kidney. But the experi-

ments proved that no lessening of the outflow amount took place either by the reflex irritation of the vaso-motor centres, or by the toxæmia of asphyxia or strychnine. In all these cases an increase of the blood-stream was regularly observed, and indeed exactly corresponding to the raised blood-pressure in each case.

This increase of the stream-velocity was particularly marked in strychnine-poisoning.

The fact that no narrowing of the cerebral vessels was produced by the irritation of sensory nerves is of special interest, because it stands opposed to the prevailing theory. It has been supposed that the swooning or convulsions following violent sensory irritation are due to cerebral anæmia brought about by reflex contraction of the brain-vessels. The authors' experiments, with intact skulls, have shown that not only no cerebral anæmia appears in those conditions, but, on the contrary, the brain is more richly permeated by blood, in consequence of the heightened blood-pressure.

Concerning the action of narcotics, the authors found that at the commencement of chloroform inhalation, the circulation in the brain is considerably accelerated, and simultaneously the arterial blood-pressure rises. After a short time, which often is less than a minute, the blood-pressure begins to sink, notwithstanding which the outflow quantity remains increased, and often amounts to three or more times the original when the blood-pressure already has sunk to, or below, the level existing before the inhalation. This acceleration of the blood-stream is thus independent of the blood-pressure, and is only to be explained by a widening of the cerebral vessels. If the chloroform inhalation be continued, the blood-pressure may become so minimal that little or no blood flows through the expanded blood-vessels. If the inhalation be stopped and the blood-pressure thereby be allowed to recover, a copious stream again begins to circulate. If the animal awake from the narcosis, there is often a divergence in the behaviour of the blood-pressure and cerebral blood-stream; while the blood-pressure rises, the flow of blood in the brain diminishes, evidently because the vessels, which had been expanded by the chloroform, contract again to their normal sizes, and thereby neutralise the accelerating influence of the heightened blood-pressure.

No similar property seems to belong to morphia. The fluctuation in the blood-stream which appeared after morphia injection ran an almost parallel course to the variations in the blood-pressure. At first a transient rise of blood-pressure was sometimes observed, and, corresponding thereto, an increase of the outflow; then the

pressure sank below normal, and concurrently the blood-circulation in the brain was slowed—again to be increased as the pressure rose.

Experiments with amyl nitrite produced a manifest expansion of the cerebral vessels, though to a less degree than chloroform.

The effect of direct electrical stimulation of the brain cortex in the motor area was not in harmony with the common theory, which is based upon Kussmaul and Jenner's researches. That theory supposes that an anæmia of the brain, consequent on contraction of the small vessels, exists during an epileptic fit, and that the loss of consciousness is due to the cerebral anæmia.

The constant result of thus directly exciting the cerebral cortex, in the authors' experiments, was a considerable acceleration of the circulation through the brain. This acceleration was usually not observed until 10·30 sec. after the commencement of the stimulation, although the latter, in most cases, was immediately followed by a considerable rise of blood-pressure. At the beginning of excitation there may have been present a slight contraction of the vessels, which delayed the appearance of the quickening of the brain-circulation corresponding to the increased pressure. This point was not fully elucidated by the researches.

The authors further ascertained that the quickened circulation, due to the cerebral irritation, may by no means be caused by the raised blood-pressure alone. It was of longer duration than the latter, and often when the blood-pressure had sunk to the normal or subnormal, the efflux had remained greatly excessive. The acceleration in the blood-stream was quite as distinctly marked in some cases, in which no increase of blood-pressure followed the electric irritation. Thus it was shown that an active hyperæmia of the brain, especially pronounced at the onset of the convulsions, results from electric stimulation of the motor area.

No effect on the cerebral circulation was caused by irritating the divided vago-sympathetics.

Spehl on the Distribution of the Blood circulating within the Encephalon (*Encéphale*, Jan. 1887).—This paper is mainly concerned with a comparison between the amount of blood contained in the head during the wakeful condition with that during chloral hypnotism.

The author indicates, that prior to 1860, the prevailing theory associated sleep with a congested state of the brain. Durham's

experiments, in which he directly observed the vascularity of the brain through a trephine aperture, then showed that the brain during sleep is relatively in an anæmic condition. Many other investigators in later times have confirmed Durham's conclusions, by repeating his mode of research, or by obtaining graphic records of the cerebral movements. On the other hand, an attempt to revive the older theory has not been wanting.

Spehl has sought to elucidate the matter by another process. After subtracting 1 cc. of blood from the carotid of a rabbit, for the preparation of the standard solution, the circulation between the head and trunk of the animal was suddenly arrested by means of an *écraseur* placed close to the head, and the latter was finally separated without the loss of any blood. The total amount of blood contained in the head, and that in the remainder of the animal, were then separately determined.

The mean of five experiments performed upon rabbits in the wakeful state gave $\frac{1}{8}$ as the relative amount of blood contained in the head to the total mass of the blood; whereas the mean of six estimates in the case of sleeping rabbits showed that the head contained only $\frac{1}{11.5}$ of the total blood.

In each series of experiments the mean weight of the bloodless head was $\frac{1}{10}$ that of the exsanguineous animal.

The author thus summarises his conclusions:—

1. During the sleep produced by chloral, the brain, taken, *en masse*, is anæmic.

2. Chloral hypnotism very closely resembling natural sleep (Labbée, Gubler, Rabuteau, Claude Bernard, Liebreich, Bouchut, &c.), it is probable that during the latter the brain is equally anæmic.

3. During sleep, natural or otherwise, certain parts of the brain are in activity, while others are in repose.

4. The active portions probably are congested; the parts in repose are physiologically anæmic.

ERNEST BIRT.

Zelgersma on the Morphogenesis of the Crus Cerebri (*Nederl. Tijdschrift voor Geneeskunde*, No. 21, 1887).—During the anatomical and microscopical examination of five cases of idiotcy, the author was struck by the great influence which the degree of development of the great hemispheres in idiots has on the evolution of the crus cerebri. A primary severe lesion of the cortex cerebri in the fœtal period or in earliest childhood,

existing during a long time, and causing secondary degeneration and inhibition of evolution in certain parts in the crus cerebri and medulla oblongata, characterised these five cases. In three cases hemiatrophia cerebri was demonstrated; in two other cases the alteration of the crus was very ev ident when compared with normal preparations. The alterations in all cases were nearly the same. They were as follows:

Atrophy of pons varolii and nuclei arciformes; in cases of hemiatrophia cerebri, the cells as well as the transverse bundles and direct fibres to the cortex, were affected on the same side. The pyramidal system was not always affected; here atrophy was only evident when the lesion of the cortex was severe and extensive. The author never saw degeneration of pyramids extend beyond the reflex centres in the medulla oblongata or spinal cord. Besides the atrophy of the pyramidal system, there was atrophy of the transverse fibres in the corpora pyramidalia near the nucleus olivaris in four cases. In all cases the transverse fibres of the pons varolii, which pass through the raphe and brachium pontis, to the cerebellar hemispheres of opposite sides, were atrophic. The nuclei arciformes, cells as well as nerve fibres, were affected. Bechterew's nucleus reticularis, as well as Meynert's bundle of fibres were atrophic. Alteration of nucleus olivaris was not constant. In four of the cases a bundle of fibres from the cerebrum to the nucleus olivaris, described by the author on a former occasion, was always atrophic. In one of the cases the tract between the olive and cerebellum through the raphe and corpus restiforme of the opposite side was atrophic. In four of the cases the cerebellar hemispheres on the opposite side to the brain were atrophic. In one case, the nucleus dentatus also was affected. In all cases the pedunculi cerebelli ad cerebrum were atrophic.

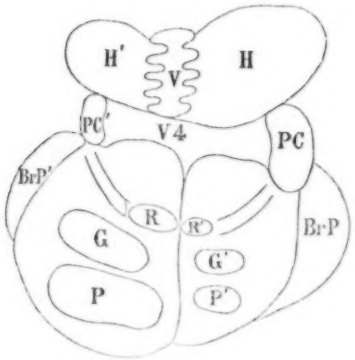
A transverse section through the pons varolii behind the posterior corpora quadrigemina shows a characteristic asymmetrical configuration, in consequence of the passage of decussated and direct, atrophic and normal fibres; this condition is represented by the diagram on page 287.

The nucleus ruber, substantia nigra, and corpus Luysii, are all atrophic. The alteration in the optic thalamus is very complicated. The anterior part is generally atrophic.

There was no affection of the corpora geniculata interna and externa, corpora quadrigemina anteriora and posteriora, and the pulvinar.

From this description, the author concludes the existence of two

different systems; one which connects the great hemispheres with the cerebellum, and the other which connects the hemispheres with the reflex arc (system of pyramids and fillet). The first connection passes through the capsula externa, pes pedunculi, pons varolii, nucleus olivaris (decussating in the raphe), brachium pontis and corpus restiforme to the cerebellum; the fibres from the pons go exclusively to the cerebellar hemispheres; those from the nucleus olivaris pass to the vermis cerebelli back. From the cortex cerebelli the path returns through the nucleus dentatus, pedunculi cerebelli ad cerebrum (decussating near the corpora quadrigemina), and the nucleus ruber, from which radiations arise to the capsula interna and optic thalamus. The difference between degeneration in this system, to which the author gives the name of



P. Pyramids.
G'. Ganglia of Pons Varolii.
R. Nucl. Reticul. pontis.
BrP. Brach. Pontis.
PC. Pedunc. cerebell. sup.
H. Cerebell. hemisphere.
V. Vermis cerebelli.
V4. Ventric. quartus.

intellectual path, and degeneration in the pyramidal and fillet system, is that the former is not governed by the Wallerian law. All these groups of cells which are found in the intellectual path are always degenerated. In the pyramidal and fillet system the degeneration ceases at the first group of cells which is met with. The author concludes that the above-mentioned ganglia are dependent on the evolution of the "intellectuorium."

These results induce the author to suppose, that there exist in the medulla oblongata and crus cerebri three systems of nervous elements: (1) the above-described intellectual tract; (2) the connection between the "intellectuorium" and the reflex arc; and (3) the reflex arc.

This supposition the author proves to be strongly supported by the history of the development of the nervous system. The

function of his "intellectual tract," the author supposes to be of a psychological nature.

The author discusses in three chapters the properties of these systems, and refers the reader for further information to a more elaborate paper in the course of preparation.

J. A. VOORTHUIS, M.D. (Amsterdam).